Spinal Injection Techniques

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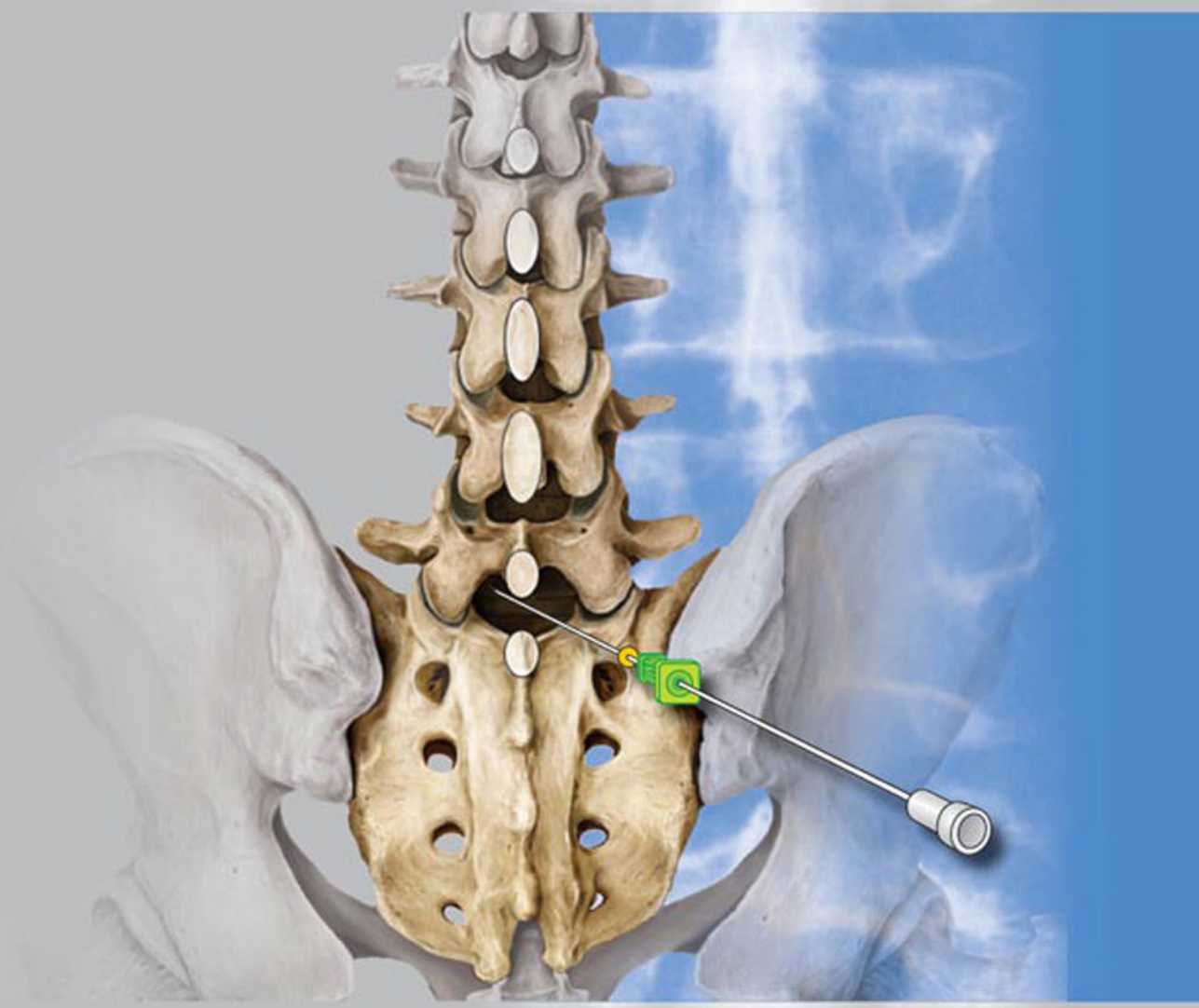

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Foreword

"The burden of the back"—back pain in all its various forms has always been and will remain the domain of the specialist field of orthopedics.

The task of "conquering" back pain, or at least implementing adequate forms of therapy, is becoming increasingly difficult under the conditions found in the Public Health Services with their lack of funding and concepts.

Treatment guidelines are strictly monitored using "evidence based medicine." This is definitely advantageous to patients and practitioners, but can also result in the freedom of treatment being "legally patronized." It is characterized by conflicts about which line of thought should be followed, especially by the societies "that argue about back treatment."

During the development of pain therapy in Germany over the last 10 years we have learnt a great deal from the conflicts relating to back pain in all of its forms. This process must be continued.

This book should aid in improving confidence when treating back pain. It is based on the work and new concepts from the "Interdisziplinären Gesellschaft für orthopädische und unfallchirugische Schmerztherapie" (Interdisciplinary Society for Orthopedic and Trauma Surgical Pain Therapy—IGOST) and represents the pain therapy section of the Deutsche Gesellschaft für Orthopädie und Orthopädische Chirurgie (German Society for Orthopedics and Orthopedic Surgery—DGOOC).

More than 90% of back pain is treated as "nonspecific" simply because the therapist knows and has learnt too little about the back. The further development of this phenomenon has to be counteracted.

It is also necessary to prevent the overuse of "technical methods and injection techniques under radiological monitoring" solely because the "traditional orthopedic clinical examination" has fallen into oblivion.

Rather, the wide range found in an established orthopedic/trauma surgical therapy should be depicted, with its proven long-term therapy success.

This book makes it clear that all spinal injection techniques were and will remain the domain of orthopedic and trauma surgery, as IGOST has conveyed in its courses.

A portion of this book is also dedicated to the rational pharmaceutical treatment of back pain, taking into account new findings in the area of local anesthetics.

The core of the matter here is the prevention of chronification of back pain using interdisciplinary therapeutic approaches and a specific early therapy management, and not simply the "management of an ever-increasing number of pain patients."

This book corresponds to the "golden standard" for spinal injection therapy from an orthopedic/trauma surgical point of view and is an excellent companion for our courses. It complements a succession of remarkable specialist books which have emerged with the help of IGOST over recent years.

It is on this premise that I thank the authors for their work with the IGOST and wish them success with this book.

I wish all our readers enjoyment and success with the teachings of this book and hope that they are able to apply what they have learnt in their daily work.

H. E. Brunner, MD
GOST–IMPS President

Preface

Spinal injection techniques enjoy a high status within pain therapy and are a valuable treatment tool when correctly used. Performing special injection techniques from the cervical spine to the sacrum professionally and safely requires a sound specialist knowledge in topographical anatomy. Building on this, the injection techniques have become increasingly refined over the years. Safe treatment for doctors and patients can be achieved when the techniques are correctly implemented. Routine radiation-charged radiographs and computer tomographies are not necessary when using the techniques described here.

Basic principles, diagnostics, causal and symptomatic pain therapy for spinal syndromes, as well as the new IRAP (EOT) method are, amongst others, extensively described in the theoretical section of the book. New concepts in multimodal pharmaceutical pain therapy are likewise demonstrated.

The atlas section conveys detailed anatomical knowledge. The injection techniques are clearly described "step-by-step" using series of photographic material. The book is divided into regions of the body for easy reference of special techniques. The diagnosis and management of therapy side effects and treatment complications are clearly arranged according to injection and area of the body.

Over 30 years of experience using injection techniques along the spine have been incorporated into this book.

We thank all of our patients who were willing to contribute to the development of this book.

A large number of the photographs were prepared by the following members of the photographic department at our clinic: Birgit Greifenberg, Silke Bachmann, and Michael Müller. It was always a pleasure to combine medicine with art. We sincerely thank you for this.

Special thanks go to the Clinical Anatomy Research and Education Center (Klinisch-anatomisches Forschungs- und Fortbildungszentrum—KAFFZ) at the Ruhr University's Anatomical Institute in Bochum, Germany. This center is an establishment dedicated to research and education in the clinical anatomy fields. In the last years the well-known "hands on" courses by the Interdisciplinary Society for Orthopedic Trauma and Surgical Pain Therapy (Interdisziplinäre Gesellschaft für Orthopädische und Unfallchirurgische Schmerztherapie—IGOST) have been held here in cooperation with the Orthopedic University Clinic. The directors of the Anatomical Institute, Professor Rolf Dermietzel, MD, and Professor Hans-Georg Mannherz, MD, and their employees, Claudia Schneider and Helmut Riese, have given us the considerable opportunity of clarifying anatomically relevant details related to the special injection techniques.

We sincerely thank the staff of Thieme Publishers for their excellent constructive input and assistance with the book and the pleasant collaboration.

We encourage all our readers to use spinal injection therapy more intensively without the use of diagnostic imaging and to implement this to their patients' advantage.

Good luck!

Theodoros Theodoridis
Juergen Kraemer

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II Atlas Section

I General Section

1 Basic Principles

Orthopedic Pain Therapy

NOTE

Orthopedics is the medical specialism that concerns itself with the musculoskeletal system.

As such, it deals with the diseases and injuries found in bones, ligaments, muscles, and joints at every stage of life. Orthopedics is described more precisely in the official definition contained in the regulations governing the continuing education of orthopedic specialists:
"Orthopedics envelops the recognition, treatment, prevention, and rehabilitation of congenital and acquired changes in form and functional disorders, disease and injury of the musculoskeletal organs, as well as the pain therapy within these areas."
The spectrum of orthopedic medicine ranges from malformations of the spine and limbs to inflammatory bone and joint diseases, pediatric orthopedics, orthopedic oncology, rehabilitative medicine, and technical orthopedics. It also includes injuries and damage to the musculoskeletal organs caused by wear and tear, as well as the pain states associated with these injuries.

The essential components of conservative orthopedics include not only the treatment of pain but also recovery from musculoskeletal disorders that affect function and form. This includes the use of bandaging, physical agents and electrotherapy, manual therapy, systemic pharmaceutical therapy, local injections, physiotherapy, and orthopedic devices.

NOTE

Pain can be defined as an unpleasant sensory and emotional experience.

The International Association for the Study of Pain has agreed on a more extensive definition of pain: "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP 1979).

This definition distinguishes pain from other unpleasant sensations by relating it to physical damage. The second part of the definition acknowledges that pain may be experienced even when no tissue damage can be demonstrated at that instant. This extension of the definition is especially important for pain that is described as chronic.

Further sensory disorders within the musculoskeletal system which possess possible warning functions include:

- **hypoesthesia:** reduced sensation to touch
- **anesthesia:** loss of sensation
- **dysesthesia/paresthesia:** abnormal sensations described as being like ants crawling, pins and needles, or a furry feeling
- **hyperesthesia:** increased sensitivity to touch stimuli
- **hyperalgesia:** increased sensitivity to pain.

These sensory disturbances occur in the musculoskeletal system with or without pain, e. g., with nerve root syndromes, peripheral nerve lesions, and in the area surrounding surgical wounds. Local numbness and sensory disorders often remain after a subsided nerve compression. They can function as an alarm, e. g., saddle anesthesia caused by cauda equina syndrome.

Acute and chronic pain are not differentiated solely by the duration of the pain. **Acute pain** in the musculoskeletal system is felt following acute events, e. g., stretching of the joint capsule, muscle tears, or disk prolapse.

NOTE

Acute pain begins suddenly, gives a warning, and elicits an immediate reaction. In most cases, this reaction involves adopting a relieving posture with an increase in muscle tension so as to combat the cause of the pain.

A **chronic pain syndrome** or **pain cronification** in the musculoskeletal system is described as pain that is constantly or intermittently present over a period of at least 3 months. The most common causes are relapsing spinal syndromes with or without radiation into the extremities. The progression from acute to chronic pain is described as chronification.

NOTE

Chronification acute pain → chronic pain.

Within the musculoskeletal system, **the chronification of pain is defined as the transition from acute to chronic pain**, where pain is present for more than 3 months and has lost

its warning function. The patient exhibits an increase in secondary psychological symptoms, with a change in the perception and processing of pain signals. The relationship between the intensity of the pain stimuli (e. g., tissue damage) and the pain reaction is lost.

The degree of cronification is dependent on:

- the duration of pain
- pain dispersion
- response to medication
- the doctor–patient relationship
- changes in experience and behavior.

Example

Symptoms of lumboischialgia persist for several months during the chronification process. The radiating pain and the area of radiation into the leg change constantly. The patient requires stronger medication to deal with the pain, and ends up by changing doctors.

NOTE

We speak of a chronic pain syndrome when the perceived pain is largely independent of the original cause of pain and has become an independent disease state.

Concomitant symptoms, such as increased muscle tension, abnormal posture, and psychogenic reactions become more important. These symptoms may even become a disease in their own right, even when the cause of pain is no longer present.

Chronic pain syndrome is also referred to as "pain disease" (Adler et al. 1989, Eggle and Hoffmann 1993, etc.) to emphasize that it is the pain itself that has become a disease. One disadvantage of this terminology is that patients are given the impression that because they have a "disease" there is nothing they can do about the pain. It is exactly this interpretation that is detrimental in chronic pain syndrome. In fact, the opposite is true, and patients should be educated in how to actively control their pain.

An example of a chronic pain syndrome

The chronic irritation of a nerve root due to a disk prolapse or a lateral spinal canal stenosis. The symptoms often remain, even when the cause of pain has been removed (e. g., by surgery). The nervous system has learnt to perceive pain (see "Moving from Acute to Chronic Pain: Nociceptor Sensitization," below).

Orthopedists use a variety of methods to treat pain. Aside from the administration of common analgesics in general pain therapy, other methods specifically related to orthopedics include:

- physical therapy
- physical agents and electrotherapy
- manual therapy
- local injections
- orthopedic technical aids
- movement programs
- surgery.

Following injury, therapy for orthopedic pain is applied directly or indirectly to the somatic source of pain, and should prevent the chronification of pain. The progression of acute pain to chronic pain and subsequently to a chronic pain syndrome should be halted right from the start. When the initial intervention is unsuccessful, or too late, treatment must increasingly take psychological aspects into consideration. Psychology and orthopedics are of equal importance in the treatment of chronic pain, chronic pain syndromes, and somatic psychogenic disorders. Purely psychogenic disorders primarily require the input of a psychologist. At the same time, orthopedic surgeons must rule out primary organic disorders and keep a look out for secondary functional disorders as required.

Nociception and Chronification

The pain process goes through several phases: from the activation of nociceptors and conduction of nociceptive impulses, to pain perception and muscular and autonomic reactions. The sequencing of painful events in the musculoskeletal system is specific. Musculoskeletal pain arises from mechanically inflammatory, thermal, or chemical stimuli that affect bones, muscles, tendons, and joint capsules. The pain caused by these noxious stimuli is perceived in the cerebrum. At the spinal level, pain stimuli are distributed for example to the muscular system and the autonomic nervous system.

The physiology of musculoskeletal nociception is based on the existence of an extensive, independent peripheral nervous system which is dedicated to the perception of peripheral musculoskeletal pain. The process shown in **Fig. 1.1** occurs when a stimulus sufficient to cause pain irritates a nociceptive system which has not been previously damaged (by being sensitized, or by the development of chronic pain).

The noxious stimuli, nociceptors, and afferent fibers combine to form the pain-creating complex. Pain signals travel from the spinal cord into the cerebral cortex, the limbic system of the brain (which affects the emotions), the anterior horn of the spinal cord (which forms part of the musculoskeletal system), and the autonomic nervous system. A multitude of transmitters, modulators, and their

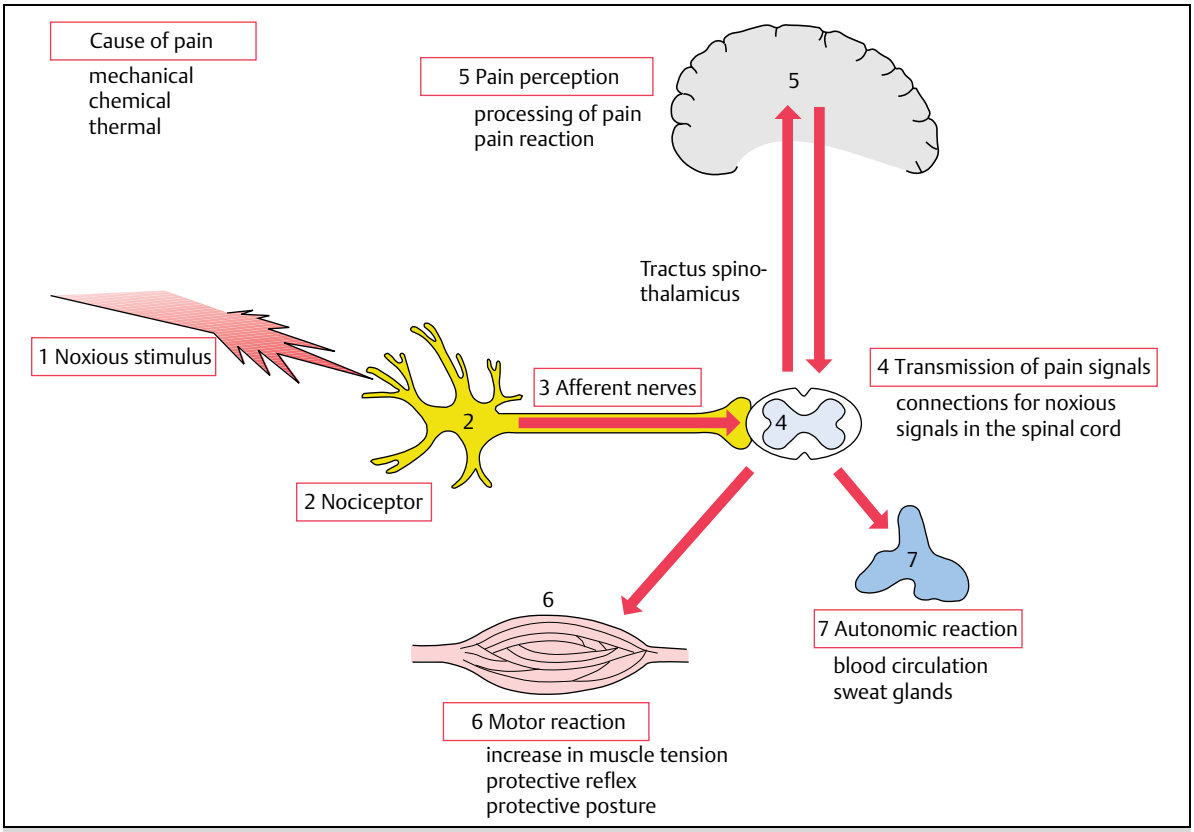


Fig. 1.1 Nociception and processing of pain signals arising from bones, muscles, tendons, and joints. The transitional areas, e. g., afferent nerves–spinal cord, are represented figuratively.

corresponding receptors participate in nociceptive processes within the central nervous system (CNS) (Schmidt and Thews 1997).

In musculoskeletal disorders, the original injury occurs in the peripheral tissues and it is here that orthopedic pain therapy acts.

Stages in the Development of Pain

Noxious stimuli (1 on **Fig. 1.1**) are events or substances that are damaging to tissues, or are a threat to them. They activate the nociceptors in bone, muscles, tendons, and joint capsules. Mechanical, chemical, and thermal exogenous stimuli act first of all on the musculoskeletal system. The direct trigger for muscular reactions as part of the vicious circle of pain and in the presence of continual psychological stress (**endogenous pain**) is unknown. Nevertheless, the process leading to the perception of pain is the same.

Nociceptors (pain receptors) (2) are nerve fibers, usually unmyelinated, that are activated when pain stimuli act on the body. The excitation threshold of the nociceptors is exceeded only by tissue-damaging irritants. The receptiveness of the musculoskeletal system to pain stimuli is dependent on the concentration of nociceptors and their thresholds. A nociceptor's response (the impulse frequency) typically corresponds to the amount of pain stimulus, increasing to the point at which tissue is damaged (Meßlinger 1997).

NOTE

Muscles, periosteum, tendons, and joint capsules contain polymodal nociceptors that react to mechanical, chemical, and thermal stimuli.

Histologically, musculoskeletal nociceptors consist mainly of noncorpuscular (free) nerve endings. They are found on the small blood and lymphatic vessels within the connective tissue spaces and on the nerves themselves, as a so-called endoneurium (Mense 1977).

NOTE

Pain sensations originating from the musculoskeletal system form the numerically largest group within the somatosensory system because the concentration of nociceptors in periosteum, ligaments, and joint capsules is high.

Pain often accompanies orthopedic disorders in these structures, which are also liable to injury, overloading, and premature wear and tear.

Afferent fibers (3): Pain signals are transmitted via afferent nerve fibers from the nociceptors to the spinal cord. Unlike dorsal column afferents, the afferent axons are thin and myelinated (Aδ-fibers) or unmyelinated (C-fibers). Visceral afferent fibers are also mostly unmyelinated.

Transmission of Nociceptive Signals

Spinal cord (4): Synapses connect the nociceptive afferents (Aδ- and C-fibers) to the neurons of the posterior horn. Impulses occurring at this switch point in the spinal cord result in the release of excitatory neurotransmitters such as L-glutamate and substance P. L-Glutamate is believed to be an important transmitter in the CNS (Zieglgänsberger and Tölle 1993). Along with other neuropeptides (e.g., substance P), L-glutamate transmits the excitatory signals from the thin nociceptive nerve fibers to the nerve cells, which in turn transmit information to the spinal cord. The nociceptive influx is transmitted from the spinal cord by the following routes, among others:

- via the spinothalamic tract to the more superiorly lying areas of the brain (limbic system and thalamus)
- to segmental neurons that are connected to the motor and autonomic reflex arcs.

The nociceptive spinal cord neurons, the so-called multireceptive neurons, receive a converging influx of signals from multiple afferents from one organ or from different organs, e.g., skin and muscle, skin and viscera. This arrangement is an important prerequisite for referred pain (Schmidt and Thews 1997). Interneurons in the posterior horn modulate the activity in these multireceptive neurons. Their activity may reduce the transmission of nociceptive signals (gate control theory; Melzak and Wall 1965).

Perception of Pain

Cortex (5): The pain impulse is transmitted to the cortex via the ascending pathways. The CNS is responsible for the integration of pain perception and the reaction to it. Parts of the pain process can be allocated to individual structures within the CNS (Zieglgängsberger and Tölle 1993).

Information about pain is integrated into the regulation of circulation and breathing within the brainstem. It is here that the descending inhibitory systems can be found. These systems play a part in the endogenous regulation of pain in the spinal cord. Inhibitory systems are constantly active in the CNS, regulating sensitivity and reaction readiness. The action of these descending and segmentally inhibitory systems can be strengthened by a variety of pain therapy methods (Zieglgänsberger and Tölle 1993). Endogenous pain inhibition is activated by electrical stimulation (TENS), morphine, afferent stimulation (acupuncture), psychological influences (stress), and movement (sport and exercise; Dietrich 2003).

NOTE

A special aspect of orthopedic pain therapy is the use of movement to reduce pain.

Motor Reaction

Muscle (6): Pain stimuli trigger motor reflexes in the form of postural adaption and protective reflexes. These actions mostly relate primarily to a change in muscle tension. Some of these reflexes are organized at the spinal level, but others are mediated by supraspinal reflex arcs (Schmidt and Thews 1997).

The change in body posture, with increased muscle tension in certain muscle groups and relaxation of other muscle groups, demonstrates a protective mechanism found in the musculoskeletal system. This mechanism aims to prevent pain stimuli irritating nociceptors. When considering orthopedic pain therapy, it makes sense **not to treat adaptive changes in posture or muscle tension for a few days when acute pain is present**, at least until the influx of pain stimuli has been disrupted and the nociceptors have been eased.

Autonomic Reaction

Autonomic nervous system (ANS) (7): The autonomic reaction to pain stimuli acting on bones, muscles, tendons, and joints is linked to a spinal reflex arc. Pain stimuli affect the ANS in different ways, depending on their origin. In the musculoskeletal system the effect is primarily in the form of a reflex increase or decrease in blood flow caused by muscular contraction: the physiological muscle pump is affected. Depending on the level of autonomic involvement, changes in skin temperature and moisture levels may occur (via the influence of sweat glands), as well as sensory disorders.

Moving from Acute to Chronic Pain: Nociceptor Sensitization

When sufficiently irritated by noxious stimuli, nociceptors send signals to the cortical areas responsible for pain perception. The processing of pain signals and a pain response follows. This process, from pain development to pain perception, can be disrupted at different levels by exogenous and endogenous factors. Exogenous stimuli and endogenous inflammatory mediators such as bradykinin, histamine, prostaglandins, and interleukins can activate nociceptors so strongly—and, what is more, so frequently—that the irritation thresholds of the nociceptors are reduced and they become more sensitive.

Pain adaptation does not occur within the musculoskeletal system. This means that it is impossible to get used to

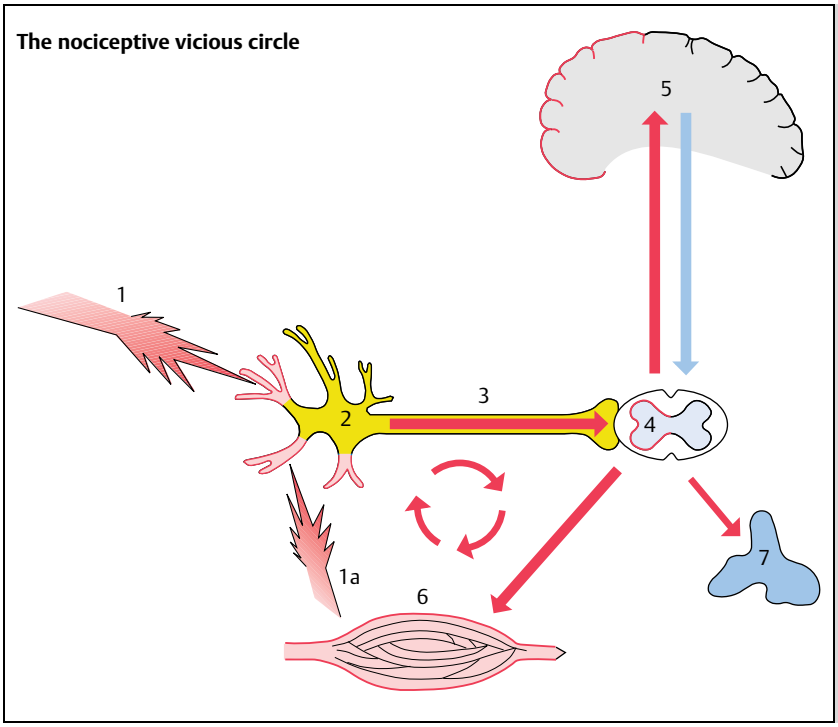


Fig. 1.2 The nociceptive vicious circle. Sensitized nociceptors (2) give rise to permanent muscle contraction (6) via the spinal reflex arc (3 and 4). The muscle spasm is in turn an endogenous stimulus (1a) for the nociceptors (2). This vicious circle can proceed independently without the influence of additional noxious stimuli (1), the participation of higher brain areas (5), or the ANS (7).

shoulder, knee, or back pain. Rather, pain threshold measurements indicate that continuous irritation results in sensitization of nociceptors (Schmidt and Thews 1997). The sensitization of nociceptors can occur with inflammatory processes resulting from a range of causes such as injuries, active arthritis, infections, or rheumatoid arthritis. At the same time macrophages are activated by the inflammatory process (e. g., via lymphokines) as part of the cellular reaction. This in turn releases antigen stimuli from the T lymphocytes. The macrophages form **prostaglandin**, **leukotrienes** and **cytokines**, which all pass on the inflammatory reaction to other cells (endothelium, fibroblasts; Zimmermann 1991). The combination of two inflammatory mediators, such as prostaglandin E2 and bradykinin, potentiates the reaction (Mense 1981).

NOTE

Sensitized nociceptors in the musculoskeletal system react to even the smallest of external influences (e. g., normal joint movement, warmth, cold, temperature change) because of their decreased irritation thresholds.

Some nociceptors become active only when inflammation is present (Schmidt and Thews 1997). These mechanoreceptors, the so-called silent or **sleeping nociceptors**, first become sensitive to mechanical input under sensitizing conditions such as inflammation (Meßlinger 1997).

Neuropeptides, including substance P, calcitonin gene-related peptide (CGRP), nitric oxide (NO), prostaglandin, and other vasoactive substances are released from irritated nociceptors (Zimmermann 1991). Nociceptive processing with sensitized nociceptors results in a vicious circle. Permanently sensitized nociceptors produce a continual change in muscle tension, as part of a protective reflex posture. This muscle tension acts in turn as an endogenous irritant to the sensitized nociceptors. Even when the exogenous irritating stimulus is no longer present, this vicious cycle remains (**Fig. 1.2**).

The orthopedic approach to pain therapy and the vicious circle of pain deals first of all with the sensitized nociceptors (2) and the afferent fibers (3), then moves on to pain transmission (4). Its primary role, however, is to influence the motor reaction by reducing muscle tension. It makes sense to make the primary intervention where the nociceptive process starts, before the vicious circle and chronification begin: with the pain stimulus itself and at the nociceptors.

Chronification by Means of Gene Activation

The ability of nerve cells to react more effectively to the same stimulus when it is applied repeatedly is nowadays seen as an important factor in the formation of memory, e. g., movements becoming more selective and economical when they are practiced. The reaction of nerve cells in the spinal cord to neuropharmacological and molecular biological techniques has been investigated; in particular, how nerve cells “remember” painful stimuli and correspondingly overreact, and how this process can be pre-

Table 1.1 The Differences between Nociceptive and Neuropathic Pain

Nociceptive pain	*Neuropathic pain*
Characteristics already present	Characteristics develop first
Local sensation of pain	Sensation of radiating pain
Site of damage and area of pain are identical	Site of damage and area of pain are different
Treatment: local anesthesia at the area of pain	Treatment: local anesthesia away from the area of pain

vented by the use of medication (Zieglgänsberger and Tölle 1993, Even 1995, Hunt et al. 1995, Tölle et al. 1995, etc.).

The activation of certain genes is important for chronification. This leads to the new synthesis of receptors and ion channels, and to stimulation or inhibition of the production of individual neurotransmitters. Molecular biology research (e.g., Even 1995, Tölle et al. 1995) demonstrates that the expression of so-called proto-oncogenes (immediate early genes, IEG) is stimulated by pain stimuli increasing neuronal excitability.

The formation of pain memory is initiated by nerve cells reacting to repeated pain stimuli with neuronal learning. A repeated stimulus of the same strength, or even weaker, causes the dorsal horn neuron to discharge, with continually increasing strength. The pain cells in the CNS react with a higher discharge frequency. Specific types of damage in the musculoskeletal system (such as strains, pressure, and nociceptor and nerve lesions) act as stimuli that can also change the phenotype of the neuron.

The task of orthopedic pain therapy is to eliminate these types of damage with causal therapy such as pain-free positioning, switching off nociception in the periphery and transmission of pain signals. This prevents or, in some cases, reverses complex adaptive processes at a morphological, neurophysiological, and genetic level.

NOTE

The chronification process can be prevented and reversed when pain that has not yet become chronic is treated with local anesthetics and an exercise program aimed at reducing pain.

Endogenous opioids that are produced by spontaneously activated spinal cord and cerebellar neurons reduce the expression of the IEG and curb the activation of nerve cells by pain stimuli. This can prevent chronification.

Neuropathic Pain

An important fact to bear in mind when addressing the symptoms of nerve root irritation and peripheral nerve compression syndromes is that damaged nerve roots and peripheral nerves discharge spontaneously. This is not the case with intact, undamaged nerves. The observations of Kuslich and Ulstrom (1990) during intervertebral disk operations under local anesthesia demonstrate this. When they touched the nerve root that had been irritated by the disk prolapse, the patient perceived the same amount of pain as when they touched the vertebral joint capsule (with its high concentration of nociceptors) and the posterior longitudinal ligament. In contrast, nerve roots from neighboring levels that had not been compressed were just as insensitive to being touched with tweezers as structures with a lower concentration of nociceptors, such as ligamenta flava, dura, and epidural fatty tissue.

Nerves become the source of pain without the primary nociceptors playing a part. Nerve fibers that are normally only responsible for relaying feelings of pain send out pain impulses. In contrast to nociceptive pain, the pain caused by the pathophysiological generation of impulses at nociceptive fibers (not at the nociceptors) is called neuralgia, neuralgic pain (Schmidt and Thews 1997), or neuropathic pain (**Table 1.1**). Neuralgic pain in the musculoskeletal system is fundamentally different from nociceptive pain. The impulse activity originating in the nerve fiber is projected into the entire area supplied by the nerve, as such impulses normally arise from the nociceptors in this area. With neuralgias, the site of the original damage is not identical to the area of pain perception. In the case of lumbar nerve root syndromes, for example, pain can be perceived in the leg via the ventral ramus (see Chapter 9).

The conversion from pain conductor to source of pain in the musculoskeletal system is a slow process. A single, short-term compression, such as the contact of an injection needle with the vertebral nerve root, or the intraoperative irritation of undamaged nerve roots (Kuslich et al. 1991) has no lasting consequences, but repeated, long-term damage results in permanent pain. Nerve pain/neuralgia develops only from long-term irritation (**Fig. 1.3**).

NOTE

The nerve becomes the source of electrical signals. The CNS gradually learns to perceive pain.

Secondary Pain

In orthopedic pain therapy, special attention should be paid to the secondary pain phenomena caused by the primary disorder or, in some cases, by the treatment itself. Fowler's position ("psoas position") or exercises involving lifting from a crouched position can provoke patella problems, for example. Insertion tendopathies can also develop as a result of unaccustomed physiotherapy exercises. Sec-

ondary pain states occur especially when reflex muscle tension increases and adaptive postures are adopted. In the case of pain that varies according to weight-bearing, asymmetrical loading on one leg leads to an asymmetry in the pelvis with irritation and chronic pain in the lumbar–iliac junctions as well as in the lumbar zygapophyseal joint capsules. The secondary pain can also become chronic, becoming a pain in its own right after the primary source of pain is no longer present.

Mixed Pain Syndrome

Generally speaking, chronic pain syndromes in the musculoskeletal system are a combination of nociceptive pain and neuralgia. The mixed radicular–pseudoradicular cervical and lumbar syndromes are typical examples of this. Alongside the dermatome-oriented neuralgias with radiation into the extremities, there is almost always local pain in the affected spinal segment. This pain comes from the nociceptors in the zygapophyseal joint capsule, the posterior anulus fibrosus, and the posterior longitudinal ligament. In addition to this, secondary pain occurs as a result of increased muscle tension and maladaptive postures, sometimes also in other sections of the musculoskeletal system. The secondary symptoms arising at the sacrococcygeal joint during a compression syndrome of the lumbar nerve roots are a typical example of this (**Fig. 1.4**).

When dealing with a lumbar nerve root syndrome, an essential prerequisite for successful orthopedic pain ther-

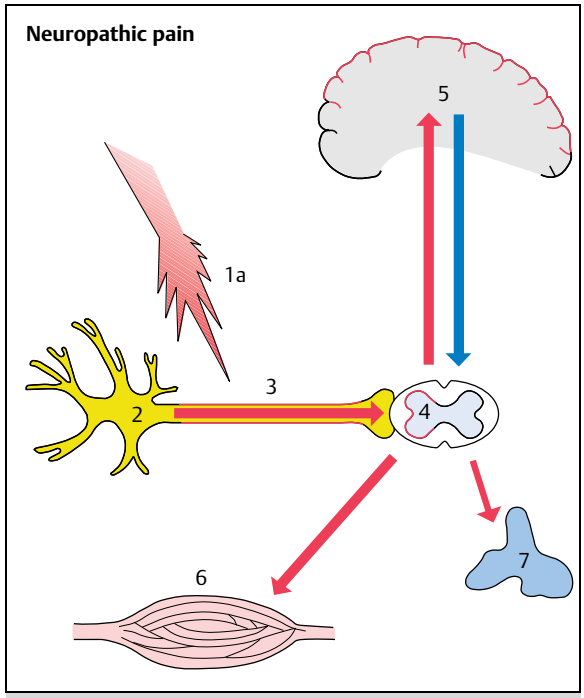


Fig. 1.3 Neuropathic pain (neuralgia). The pain stimulus (1a) acts directly on the nerve (3). The nerve becomes the source of pain without the nociceptors (2) playing a role. The transmission of pain signals (4), pain perception (5), as well as motor (6) and autonomic (7) reactions are the same as in nociceptive pain: the nerve becomes a nociceptor.

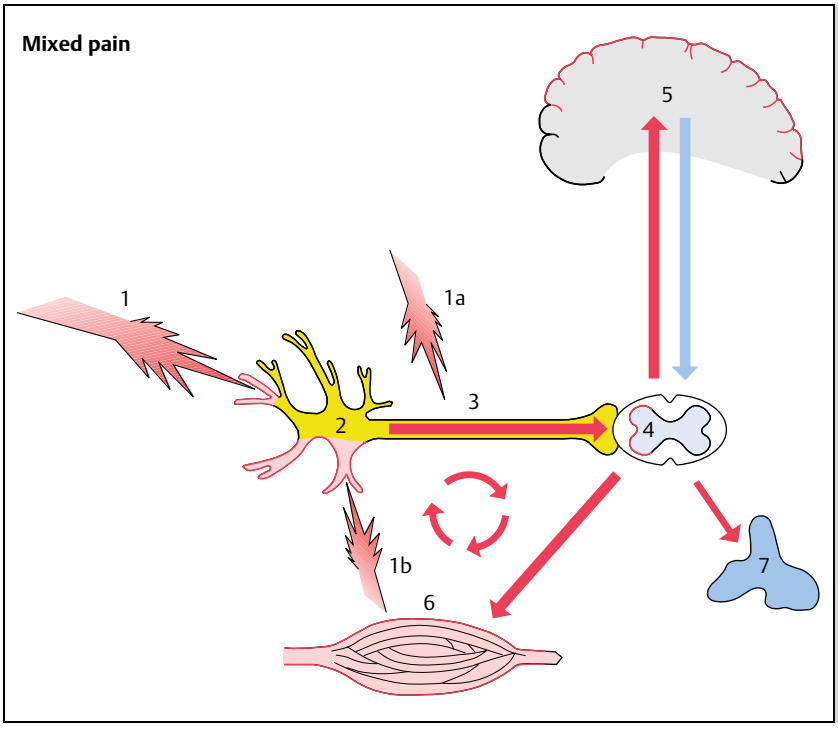


Fig. 1.4 Mixed pain: Nociceptor pain (1 and 2), neuralgia (1a and 3), secondary pain (1b and 6).

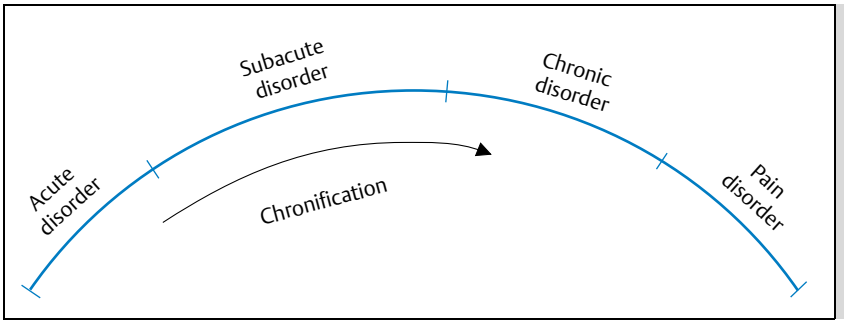


Fig. 1.5 The pain spectrum ranges from acute via subacute to chronic. Chronic pain syndrome represents an extreme form of chronification.

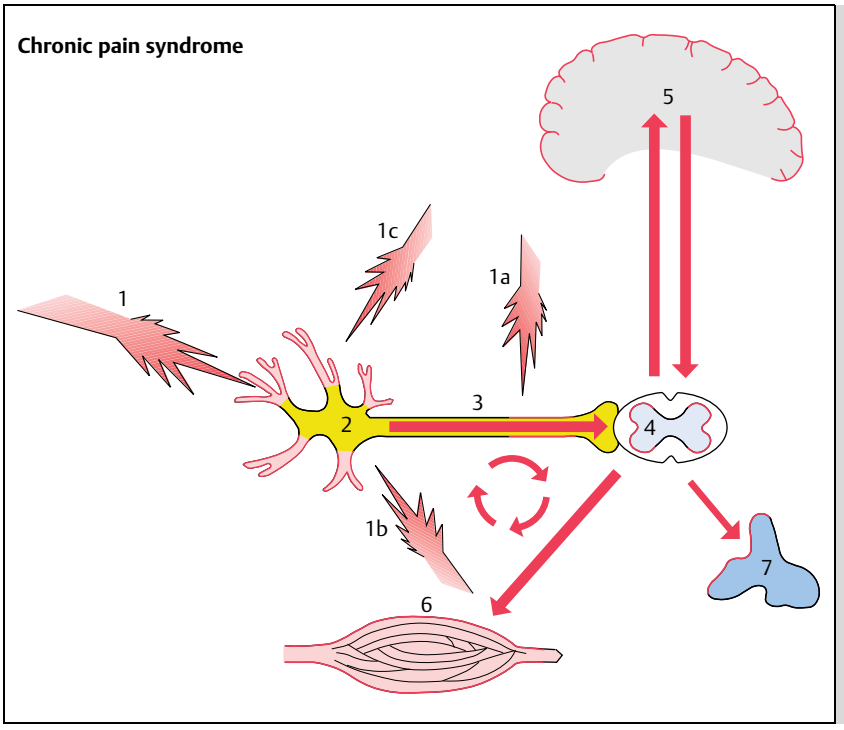


Fig. 1.6 Chronic pain syndrome (pain disorder) in the musculoskeletal system. Nociceptive cycles in the musculoskeletal system have become independent. For example, when signals are transmitted from nociceptors (2) or afferent fibers (3) to the brain (5) via the spinal cord (4) they are then transmitted as endogenous stimuli (1c) back to the nociceptor (2) or to the afferent fibers (3). A further cycle is formed via the spinal reflex arc. Signals are transmitted to the muscles, which tighten up and act as endogenous stimuli (1b) to the nociceptor (2) or the afferent fibers. To maintain this cycle, peripheral nociceptive input must be present in the form of exogenous stimuli (1) for the nociceptors (2) or as stimuli (1a) for the afferent fibers (3).

apy is the analysis of the individual pain components, using, e. g.,

1. clinical neurological assessment of the root syndrome
2. assessment of the sacroiliac and the zygapophyseal joints using manual techniques
3. postural and loading analysis
4. assessment of the emotions.

The orthopedic pain therapy approach therefore has different aspects too, using, e. g.:

re 1: local treatment of the nerve root compression with positioning to relieve compression using orthopedic aids, local injections, general measures
re 2: traction in the pain-free direction
re 3: positioning for relief, heel raise, posture and behavioral training (back school)
re 4: relaxation exercises, methods of coping with pain so as to activate central pain inhibiting systems, reduction of central nervous motor arousal.

Chronic Pain Syndrome

A pain disorder is an independent disorder that develops when pain is no longer associated with its original source. Chronic pain syndrome is an extreme form of chronification (**Fig. 1.5**). Nociception in different areas of the musculoskeletal system is affected at the same time. Numerous circuits develop, and associated symptoms become a priority. The moment when the original pain started loses its significance. Pain perception, pain processing, pain reaction, and pain transmission form their own cycle.

In addition to the normal process of pain stimuli/nociceptor/afferent fibers, transmission of pain perception, motor and autonomic reactions, there is also a pathway running from the brain (5 on **Fig. 1.6**) directly to the nociceptor (2) or to the afferent fibers (1c). This means that the vicious circle of pain can be stimulated by centrally governed areas. Afferent fibers are stimulated, pain signals are distributed at a spinal level and result in motor reactions,

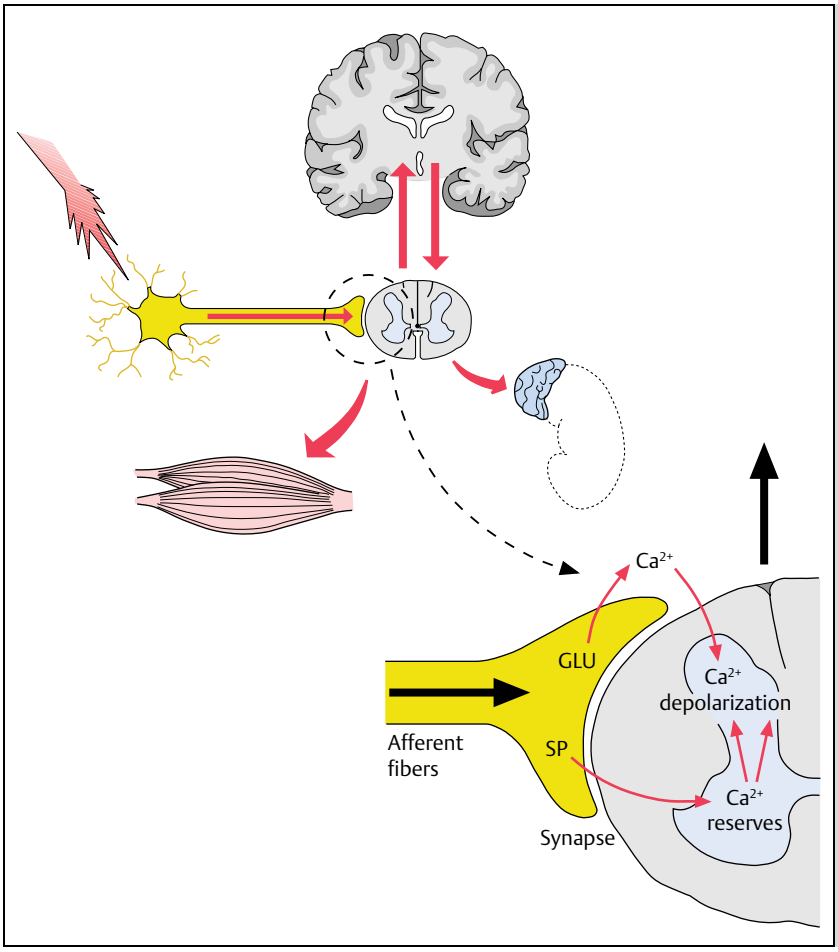


Fig. 1.7 The learning processes in pain-processing nerve cells (Zieglsgängsberger, Tölle 1993). Pain impulses are transmitted along afferent fibers to the synapses in the spinal cord. It is here that L-glutamate (GLU) and substance P (SP) are released. L-Glutamate facilitates the influx of calcium ions (Ca^{2+}) into the nerve cells. Substance P releases calcium ions from the intracellular reserves. A higher number of calcium ions in a nerve cell increases its excitability, resulting in increased transmission to the brain. The more frequently this occurs, the more quickly the nerve cell discharges when a pain stimulus is present.

and nociceptors or neuropathically altered afferent fibers are irritated.

The **learning processes** found within nerve cells play an important role in the development of chronic pain syndromes. When a pain stimulus is applied repeatedly, nerve cells discharge more frequently and eventually start to discharge spontaneously (Zieglgänsberger 1986). Processes are stimulated in the nerve cells that make it possible for the cell to convert brief synaptic periods of irritation (e.g., from a pain irritant) into a long-term adaptive change, taking part in the creation of the nerve cell's "pain memory." Calcium ions flow into the internal part of the cell or are released from intracellular reservoirs when the nerve cell is activated by transmitters such as L-glutamate or substance P.

The reactivity of nerve cells changes when permanent or frequently recurring pain is present. The increased regeneration of different proteins and peptides such as receptors and ion channels is a result of this (**Fig. 1.7**). When cells are no longer being prompted by continual activation, it takes some time for this altered production to be forgotten (Zieglgängsberger 1986).

Pain disorders in the musculoskeletal system always require a nociceptive stimulus to maintain the cycle of pain disorder. The exogenous nociceptive stimulus (**Fig. 1.6**, 1 and 1a) may be minimal, but it can still initiate a global reaction in a sensitized system. The **orthopedic pain therapy approach** is as follows:

1. Deactivation of independent pain cycles using appropriate medication, psychological pain therapy, and physical measures.
2. Removal of exogenous pain stimuli and nociception from the periphery, no matter how minor they may be.

Therapeutic measures include: local anesthesia, local anti-inflammatories, and causal orthopedic pain therapy.

NOTE

The somatic component of nociceptive input from the periphery should always be treated when a patient presents with chronic pain syndromes of the musculoskeletal system.

Table 1.2 Prerequisites for the Development of Chronic Pain in the Musculoskeletal System

Chronic recurring irritation of nociceptors and/or afferent fibers
Increased pain perception
Pain processing
Pain expression
Disposition
Further nociceptive input from the periphery

Chronic shoulder, neck, back, or joint pain presenting as a chronic pain syndrome is always linked to an organic structure, but the amount of perceived pain is disproportional. When the primary symptoms have been minimized (as a result of either a spontaneous remission or medical treatment), it is important to pay attention to the secondary disorders of increased muscle tension, joint malpositioning, and postural changes.

In order for a chronic pain syndrome to develop within the musculoskeletal system, chronic recurring irritation of nociceptors (2) and/or the afferent fibers (**Fig. 1.6**, 3) has to be present initially. Increased pain awareness, pain processing, and pain expression in the CNS add to this. The endogenous inhibitory systems are largely switched off. This means that minimal irritation from the periphery, whether physiological or pathological, is exaggeratedly perceived and processed, resulting in an overreaction in pain expression. To develop such pathological processes, a certain type of disposition is required. In other words, a certain habitus, neural constitution, and metabolic state are involved in the development of a musculoskeletal chronic pain syndrome. But ultimately, a constant nociceptive input from the periphery is needed to maintain the neuronal cycle in the nervous system (**Table 1.2**).

The prerequisites for the development of the chronic pain syndrome are found mostly in the mobile cervical and lumbar segments. It is here that chronic recurring irritation of nociceptors in the zygapophyseal joint capsules and in the afferent fibers of the spinal nerve takes place. The irritation occurs over a long period of time, with varying intensity. When the required predisposition is present, increased awareness of pain, pain processing, and pain expression come about. The further peripheral nociceptive input consists of the repeated, sometimes quite minimal, irritation of nociceptors and afferent fibers during normal movement.

Pain therapy consists of the complete disruption of nociception from the periphery, e. g., by repeated administration of local anesthesics.

NOTE

In the case of chronic pain syndromes in the musculoskeletal system, the causal orthopedic pain therapy reduces the nociceptive input from the periphery and may even eliminate it.

It is essential that psychological treatment accompanies the healing process when dealing with pain disorders in the musculoskeletal system.

2 Diagnostics

Medical History

Taking a precise medical history is important in the diagnosis of orthopedic pain. Acute pain originating from the musculoskeletal system demonstrates certain characteristics:

1. **It is position-dependent**, i. e., pain increases or decreases according to posture or positioning.
2. **It is load-dependent**, i. e., pain generally increases when pressure is exerted on the affected body part, e. g., when walking, standing, lifting, or carrying.
3. **It is limited to a specific area**, i. e., the patient is able to describe where the pain comes from and the area of spread.

The possible locations of source pain in the shoulder, anterior knee, neck, and lumbosacral region are densely packed together. Each location exhibits special clinical symptoms which require a special type of pain therapy. Diagnoses such as knee, shoulder, or back pain are too general and allow general pain therapy at the most.

When diagnosing orthopedic pain, the physician should ask specific questions if the patient does not spontaneously offer information. The four "-ions" have been tried and tested for this purpose:

NOTE

The four "-ions": Location—Duration—Provocation—Description

Location: Where exactly is the pain located when it occurs? It is best to let the patient show where the pain is coming from, or where it radiates to, by pointing with their finger. Nonspecific information about inconsistent, diffuse, or sock-like spread and cramp-like pain is less characteristic of musculoskeletal disorders.

Duration: How long has the pain been present? Days, weeks, years? How did it start? How has it been treated previously?

Provocation: When does the pain appear? The patient should be asked about the effects of position and weight-bearing, and also about when the pain appears: during the day, at night, mainly when sitting, standing, when walking, etc. Special clinical symptoms require special questions, such as the abduction phenomena in the shoulder or crouching low with posterior meniscal horn damage. What can be done to relieve the pain? Warmth, cold, flexion, extension, sitting, walking? How does the patient react when the pain occurs?

Examples

Patients with back pain and sciatica prefer to walk around.

Patients with lumbar spinal canal stenoses flex when standing or sitting down.

Patients with neck symptoms caused by a cervical syndrome prefer a warm shower.

Description: This relates to the quality and quantity of the pain. The use of a visual analogue scale to assess the quantity of pain has been tried and tested. It ranges from pain free = 0 to worst pain imaginable = 10, see Chapter 4, "Multimodal Medication Concomitant Therapy" (**Fig. 4.16a–c).**

Words are suggested to the patient to assess the quality of the pain. Musculoskeletal pain is most likely to be

- stabbing
- shooting
- burning.

Patients suffering from the usual acute orthopedic disorders tend to be in good **general condition**. Apart from the local problem, e. g., in the lower lumbar region, they are usually physically and psychologically healthy, provided they have not already taken too much medication. Symptoms such as nausea, vomiting, loss of weight, loss of appetite, or general feelings of weakness are not characteristic of disorders or injuries of the musculoskeletal system. If the patient reports symptoms of this nature in the subjective assessment, differential diagnoses should be kept in mind.

Patients suffering from **chronic pain** should be questioned about how and when it started. Many patients can specify the exact day and hour when their pain began. When the primary opportunity for treatment has been missed and the pain has been present for weeks or months, the characteristics of the pain may change. Localized pain becomes diffuse; the intensity of the pain is no longer position-dependent but rather becomes a permanent fixture; and the patient's general condition increasingly suf-

fers from lack of sleep, intoxication from medication, and psychosocial stress. For these reasons the details of all previous therapy have to be ascertained, including which physician has been consulted and why the treatment was discontinued. In order to treat patients appropriately, all details of their previous and current pain have to be established. The physician has to become a **medical history fanatic** when dealing with musculoskeletal pain.

Clinical Examination

NOTE
The clinical examination used to diagnose musculoskeletal pain always looks at the entire orthopedic picture. It includes a neurological assessment and specialized manual medicine techniques.

Examination of the entire body is **always** required, even when pain is concentrated in a specific part of the body (**Table 2.1).**

Example
Persistent symptoms coming from the inferior zygapophyseal and sacroiliac joints (summarized as treatment-resistant low back pain) may originate from the first metatarsophalangeal joint. A hallux rigidus is found during the examination of the entire body. This movement disorder affects the gait pattern and is suspected of causing the pain. It is therefore the primary area to be treated.

Assessment of the entire orthopedic picture consists of
- visual assessment
- palpation
- assessment of movement.

Visual Assessment
Musculoskeletal pain causes characteristic postural and behavioral changes. Patients suffering from hip and knee pain limp when walking, and patients suffering from sacroiliac joint pain or sciatica tend to have a typical asymmetrical gait. It is important to observe patients when they walk around the examination room, while dressing and undressing, and also when they climb up onto the examination couch.

Table 2.1 Orthopedic Pain Diagnostics (Clinical Examination Findings)

Entire orthopedic picture
Neurological examination
Manual medical assessment

Palpation
The typical painful pressure points are assessed during palpation. These points do not always correspond to the source of pain. It is best to let the patient point to the main location of pain, e. g., a specific spinous process. In case it is helpful for further diagnostics, the point is immediately marked with a pen and infiltrated with a local anesthetic following the clinical examination. When this trial treatment results in freedom from pain, the approach required for orthopedic pain therapy has been found.

Example
A localized cervical syndrome with a pressure point on the superomedial edge of the scapula.

Example
Baastrup disease with a pressure point between the inferior lumbar spinous processes.

Assessment of Movement
The range of movement in the musculoskeletal system is assessed and documented using the neutral-zero method. Chronic pain in a spinal segment or in a joint leads to permanent adaptive changes in posture, joint capsule contraction, and muscle contractures, and always leads to a limitation in range of movement. In order to document the pain situation objectively, these changes must be identified. Manual medicine techniques are used on the spine to diagnose limitations in mobility or complete blockages in individual segments (**Figs. 2.1**–**2.5**). The assessment of entire spinal segments is performed using the neutral-zero method.

To diagnose and monitor the progression of chronic spinal pain disorders, it is extremely important to examine and document range of movement during the pain therapy. This especially applies in the evaluation of individual pain therapy measures. The assessment of movement shows us which directions of movement are pain-free and which directions provoke pain within a spinal segment, indicating the approach that should be used in causal orthopedic pain therapy. For example, when treating limitations in mobility, pain therapy should always treat in the pain-free range of movement (Dietrich 2003).

Fig. 2.1 Segmental mobility assessment in the lumbar spine during rotation to the right.

Fig. 2.2 Segmental mobility assessment in the thoracic spine during rotation to the right.

Fig. 2.3 Initial position when assessing O/C1 rotation.

Fig. 2.4 Final position when assessing O/C1 rotation.

Example
Chronically recurring **cervical syndrome**. Cervical lateral flexion and rotation in a certain direction is usually found to be less painful during the manual medical assessment. This should be used as the starting position for Glisson kyphosis traction and movement therapy.

The manual medical examination (**Table 2.1**) is used for the fine diagnosis of functional vertebral disorders. When diagnosing pain, it is important to identify whether pain begins at the start of a movement, during it, or at the end. Pain occurring at the start of a movement indicates the presence of either inflammatory changes or a chronic degenerative pain syndrome. Pain appearing at the end of a movement implies the presence of mechanical obstructions (such as in the first phase of degenerative changes).

The same starting position should always be used when assessing mobility, and with each individual joint and spinal segment. In this way pain criteria can be established.

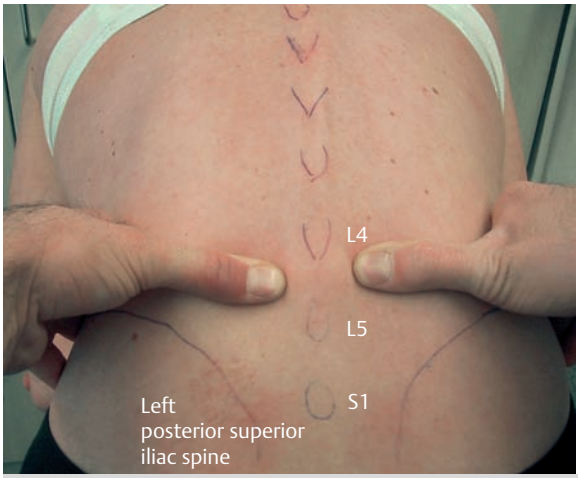


Fig. 2.5 Assessing the overtake (Vorlauf) phenomenon in the lumbar spine (positive Vorlauf phenomenon at right L4).

Neurological–Orthopedic Examination

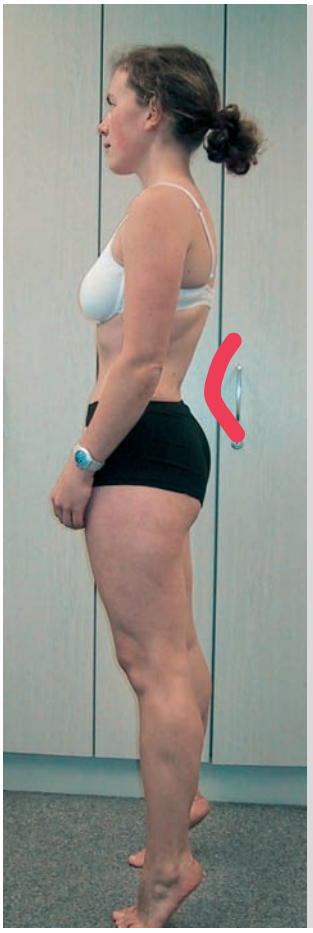


Fig. 2.6 An increase in lordosis when walking on the toes: a possible spinal narrowing is intensified when walking on the toes.

NOTE

The neurological examination is an essential component of spinal pain diagnostics.

The reflex status is examined after the visual assessment, palpation, and the assessment of movement. It includes the examination of muscle strength (**Figs. 2.6, 2.7**), sensation (**Fig. 2.8**), and coordination.

The musculoskeletal system and the neurological system must be assessed together, because of their immediate proximity and reciprocal influence on each other. For example, the visual assessment, palpation, and examination of lower limb joint range are immediately followed by the examination of the patellar tendon, Achilles tendon, and tibialis posterior tendon reflexes, as well as sensory and coordination testing and special nerve tension tests (**Fig. 2.9**).

Examination of spinal cord function is always conducted alongside the orthopedic examination of the spine. In the case of nerve root or peripheral nerve compression, the extent of nerve injury is determined by assessing the motor function (muscle strength), sensation, and reflex activity in detail. Sensory disorders, muscle weakness, and a reduction in reflex strength may be found, depending on the duration and extent of nerve compression. Conservative or surgical treatment is indicated, depending mainly on neurological deficits rather than limitations in movement.

Using the information gathered about the pain itself and the results of the neurological examination, it is pos-

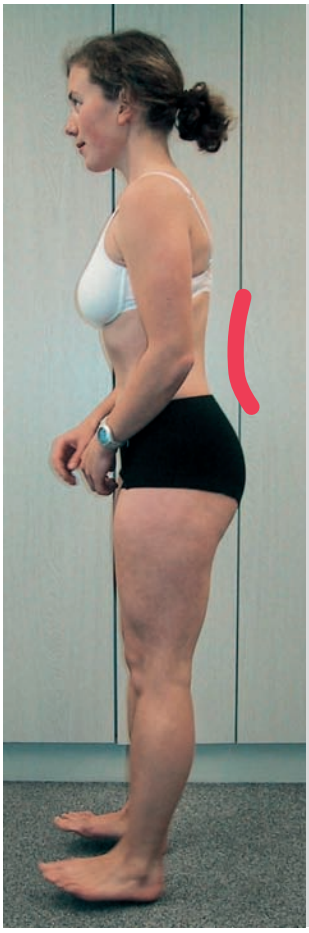


Fig. 2.7 Flattening of the lordosis when walking on the heels: the symptoms arising from spinal narrowing and/or spondyloarthrosis are reduced as the lumbar lordosis is lessened when walking on the heels.

Fig. 2.8 Examination of sensory deficits bilaterally on the lateral edge of the feet.

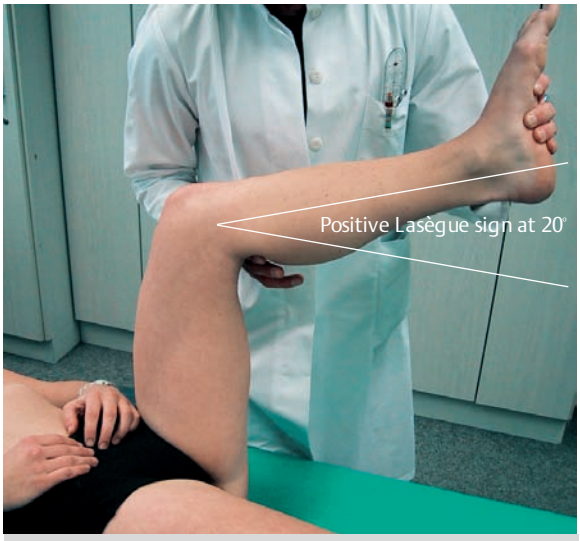


Fig. 2.9 This starting position is used for the assessment of hip mobility (internal and external rotation). This is easily followed up with the Lasègue test, assessing sciatic nerve mobility. In this case, radiating symptoms into the left leg occurred after approximately 20° passive knee extension.

sible to establish whether pain is nociceptive, neuralgic, or a mixed pain syndrome. The methods used for pain therapy correspond to this diagnosis.

Laboratory Tests

The most important serological tests are the examination of white blood cells (leukocytes) and C-reactive protein (CRP). CRP reacts quickly and is therefore especially suitable for the recognition of inflammation in its early stages. The monitoring of CRP values over a period of time is especially valuable when inflammatory bone disorders are present. The serological tests used to clarify the presence of rheumatic diseases include rheumatoid factor, antinuclear factor, antibodies for microbial antigens, and the HLA-B27 antigen. Calcium, phosphorus, and alkaline phosphatase are assessed to aid in the differential diagnosis of systemic bone disorders (hyperparathyroidism, rickets, osteomalacia, Paget disease). Metabolic disorders that are particularly relevant to orthopedics are the triad of hyperuricemia, diabetes mellitus, and hypercholesterolemia (Niethard and Pfeil 2003).

Imaging Techniques

The diagnosis should be complete before orthopedic pain therapy is started. In addition to the clinical and laboratory examinations, **imaging** of the affected area, usually radiography, should always be carried out. The source of pain can occasionally be ascertained in this way. An example is the narrowing of an intervertebral foramen caused by osteophytic reactions radiating from the uncinate process in the cervical spine. Further imaging techniques include sonography, radiological tomography, computed tomography (CT), nuclear magnetic resonance imaging (MRI), and scintigraphy.

NOTE
Imaging techniques do not form the sole basis for the diagnosis or treatment of pain in the area of the spine.

Some imaging techniques, such as spinal discography and radiculography, have become important in the diagnosis of pain when used simultaneously with local anesthetic or pain provocation.

Trial Measures for the Diagnosis of Pain

Most nonoperative orthopedic treatment methods are not particularly invasive, risky, or costly. For this reason, the use of these methods in the diagnosis of skeletal pain is recommended.

Trial injections of a local anesthetic into the main site of pain result in rapid diagnosis and therapy. Small volumes should be injected, so that the position of the site can be more exactly established. Steroids can then be added to the local therapeutic infiltration. Trial injections with local anesthetics are used on the spine:

- as zygapophysial joint injections (facet infiltration)
- as nerve root block (radiculography)
- as intradiscal injections with the provocation of discogenic pain.

The attempt to immobilize or to limit mobility in painful spinal segments not only aids the pain diagnosis, but also provides important clues for causal orthopedic pain therapy.

Example
If the trial use of a cervical collar for posterior headache reduces pain, a cervical origin is indicated.

Example
In postdiscectomy syndromes, a single-hip spica cast encasing the thigh on the painful side is used to assess the appropriateness of a spinal fusion operation.

Example
Fowler's position relieves leg pain caused by nerve root compression and spinal canal stenosis. Leg pain that is caused by arterial circulatory disorders tends to increase when the legs are raised.

Questioning patients about how warmth, cold, or vibration affect their pain provides important information for diagnosis and therapy. If the patient has not yet tried any of these interventions, they should be encouraged to do so. Generally speaking, cold relieves acute pain and heat relieves chronic pain. If the application of heat results in a dramatic increase in pain levels, the presence of bacterial inflammation should be investigated. The use of ultrasound, electrotherapy, and interferential therapy is trialed in a similar manner.

At present there is no way of diagnosing pain instrumentally. It is possible to measure the function of damaged nerves using EMG and evoked potentials, but these values tell us very little about the patient's perceived pain. For example, a seriously compromised nerve may be damaged to such an extent that the pain stimulus is no longer transmitted, even though the EMG and evoked potentials demonstrate massive pathological values. Conductivity should still be tested in the case of peripheral nerve disorders, however, especially when investigating the progression of the disorder. **The indication for surgical decompression is essentially based on the duration and severity of nerve damage, and whether remission following decompression is thought to be possible.**

3 Causal Orthopedic Pain Therapy

The methods used in the causal treatment of spinal pain are basically the same as those used in conservative orthopedic treatment, but with a different emphasis. The primary aim of causal pain treatment is to reduce pain stimuli, reducing nociception or preventing it altogether in the bones, muscles, and joints (**Fig. 3.1, Table 3.1**). Initial treatment relies on methods that are immediately available, such as positioning, orthopedic aids, and manual therapy. These are followed up by long-term measures such as postural and behavioral training (back school), physical therapy, and an exercise program.

Table 3.1 Causal Orthopedic Pain Therapy

Positioning, traction
Orthopedic aids
Manual therapy
Physical therapy
Postural and behavioral training; back school

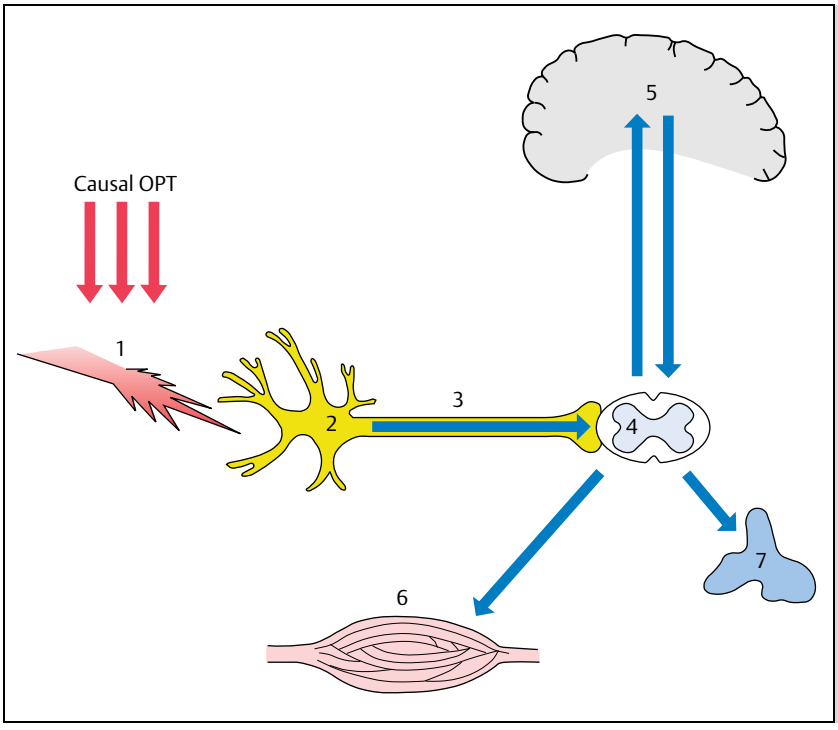


Fig. 3.1 Causal orthopedic pain therapy (OPT): Site of action, reduction, or neutralization of noxious stimuli that initiate or maintain nociception.

Positioning and Traction

Each painful section of the skeleton has a certain position that minimizes the amount of pain. The position-dependence of the pain is therefore an important diagnostic criterion.

Example
Pain originating in the spine is generally relieved when the affected spinal segment is flexed. Flexion (increasing the kyphosis) causes the intervertebral foramen to open up, relaxes the vertebral joint capsule, and flattens out disk protrusions, in every segment of the spine (see Chapter 5).
When lying supine or on one side, the patient can attain this position by flexing the knees and hips. When standing, the trunk and neck are flexed and a flexion orthosis may be used. A sitting position requires the upper body to be supported and the knees placed lower or higher, depending on the body's posture.

The spine can be further relieved when positioning is combined with the use of traction. As well as the therapeutic forms of traction usually carried out by physiotherapists, patients can be given helpful information about self-traction, the use of gait aids, or traction braces for the lumbar spine.

Orthopedic Aids

The temporary or permanent use of orthopedic aids plays a significant role in relieving acute and chronic pain in the spine and the musculoskeletal system generally, and reduces the consumption of analgesics. Orthopedic aids include splints, braces, and shoe adjustments, as well as positioning aids such as an adapted foam cube to rest the legs on when lying supine or Glisson traction on the cervical spine. Within the scope of orthopedic pain therapy, orthopedic aids are used for:

- **Immobilization,** partially or fully limiting spinal movement. The amount of movement available in spinal segments can be limited to the range that causes the least amount of pain, and ranges that provoke pain can be blocked.
- **Reduction of weight-bearing:** Reducing the load applied to the damaged part of the musculoskeletal system has a long-term influence on chronic pain.

Aids that limit painful ranges of movement and also reduce the load on damaged parts include the various types of trunk orthoses used to reduce pain in the presence of conditions such as osteoporotic compression fractures of the vertebrae, disk protrusion, spondylitis, or tumors. The use of correctly fitted trunk orthotics dramatically reduces the consumption of analgesics in these sometimes very painful disorders.

Manual Therapy

In manual therapy the hands are used to treat reversible painful functional musculoskeletal disorders. Painful hypo- or hypermobility in individual joints or groups of joints is noted by the practitioner. According to the results of the assessment, joints can then be either mobilized or stabilized by the use of exercises.

NOTE
The return to normal function using manual therapy is an example of causal pain therapy.

The aim of **mobilization** is to regain the original mobility of a joint using slow movements and large-amplitude oscillations. Joint **manipulation**, on the other hand, involves first of all moving the joint as far as possible within the pain-free range, then delivering a quick, short impulse to overcome the barriers without traumatizing tissue. A cracking sound may be heard.

The analgesic effect of manual therapy mobilization and manipulation results from the easing of joint capsule tension. This eliminates the cause of pain. Treating joint malpositioning and painful capsular tension with medication alone is purely symptomatic and temporary.

Physical Therapy

Physical therapy is a form of movement therapy that requires a doctor's referral in some countries. Special assessment and treatment techniques are used for the treatment of developmental disorders, injuries, secondary disorders arising from injuries, and disorders affecting organic and psychological functioning. Physical therapy essentially deals with healing through movement.

Physical therapy and movement therapy are used to facilitate the healing process in joints, muscles, tendons, ligaments, and bones. Generally speaking, this form of therapy addresses pain indirectly.

Physical therapy works causally by muscle strengthening and the reduction of muscular imbalances. Chronic pain originates mainly in the inferior cervical spine and the lumbar spine, but can also come from the muscular attachments in the pelvis and in the anterior knee. It is wise to treat these muscular imbalances with abdominal muscle strengthening, stretching of the hamstrings, and exercises, rather than with pain medication. Analgesics are at best used initially so that the patient is willing to do the exercises.

There are many reasons for the use of physical therapy in **symptomatic pain therapy.** Specific movements ensure the removal of inflammatory mediators surrounding the nociceptors and the nerves that have developed into nociceptors. Movement stimulates the blood flow and helps to reduce autonomic reactions and muscle spasms (**Fig. 3.2**).

Different forms of movement therapy have been developed to address orthopedic pain symptoms that involve high muscle tension and excessive adaptive postures. They utilize the body's own reflexes and the physiological behavior of nerves and muscles. In **proprioceptive neuromuscular facilitation (PNF)** the patient follows a specific pat-

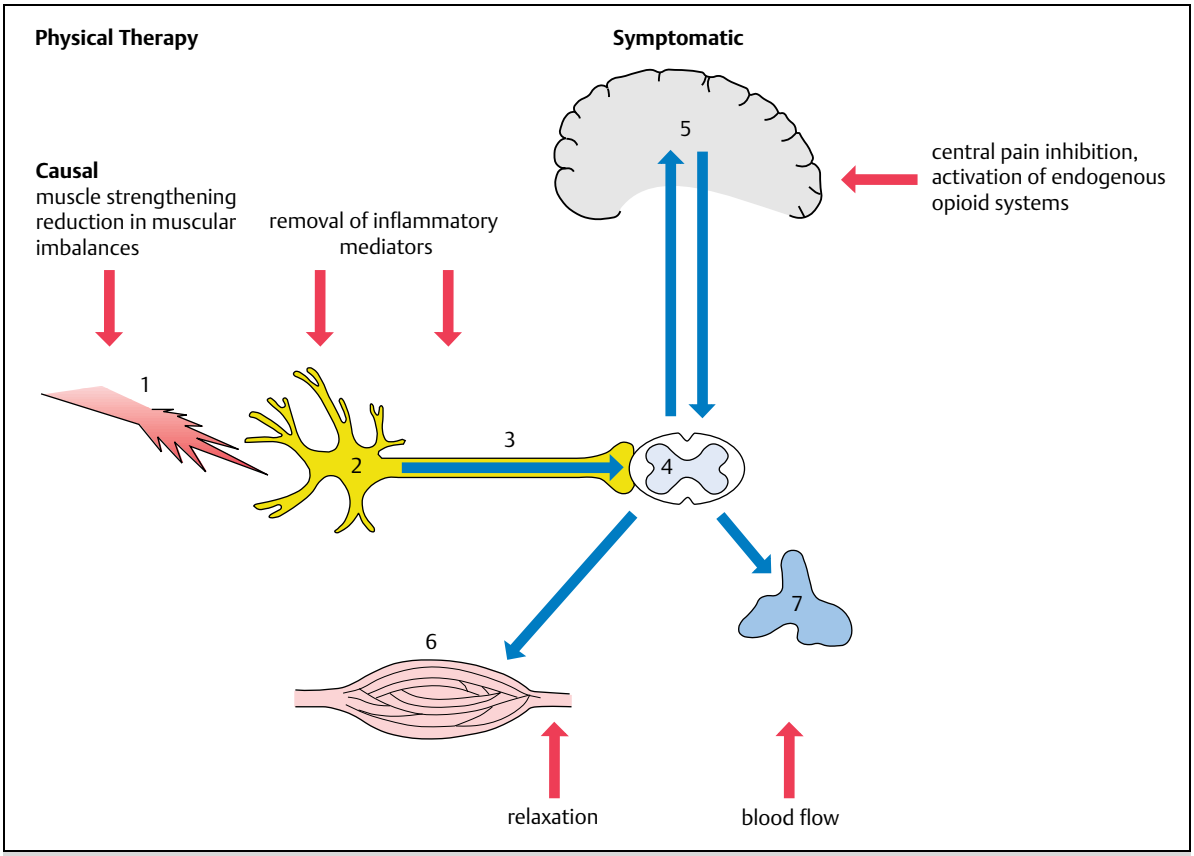


Fig. 3.2 Physical therapy and nociception in the musculoskeletal system. Causal: muscle strengthening and the reduction of muscular imbalances for the reduction (therapy) and prevention of pain stimuli affecting nociception. Symptomatic: movement therapy results in the removal of inflammatory mediators surrounding the nociceptors, afferent fibers, and muscle spindles. The movement affects the autonomic reactions positively. Movement inhibits the nervous system's nociceptive reaction and may even minimize perceived pain (5) as a result of the release of endorphins and other substances.

tern of movement against a suitably adjusted resistance while the proprioceptors are stimulated. As a result, the muscular response increases in the presence of muscle imbalances .

It is the orthopedic pain therapist's job to recognize muscle insufficiency, abnormal posture, and muscular imbalances, and to refer patients to physical therapy. Physical therapy is used with varying levels of intensity throughout the entire process of orthopedic pain therapy. When the patient's pain starts to subside, the role of physical therapy changes: it is no longer pain therapy, but a preventive program.

Postural and Behavioral Training; Back School

Table 3.2 The 10 Rules of Back School

1	Keep moving.
2	Keep your back straight.
3	Crouch down, rather than bending down.
4	Do not lift heavy objects.
5	If you must, spread the weight and hold it close to your body.
6	Keep your back straight when sitting, support your upper body, and change your position frequently.
7	Do not stand with legs straight.
8	Bend your knees when lying down.
9	Exercise, preferably swimming, jogging, or cycling.
10	Train your back muscles daily.

During the rehabilitation and prevention of spinal disorders, in particular painful cervical and lumbar spine syndromes, patients learn how to ease their back while doing physical therapy exercises. In back school, the physiotherapist demonstrates the correct ways to lift and carry weights, bend, sit, stand, and lie down. In addition, spine-friendly versions of the movements and postures needed in daily life (e. g., for dressing and undressing, bathing, or domestic tasks) are demonstrated and practiced.

NOTE

When posture and behavior are appropriate, the frequency and intensity of pain stimuli acting on the nociception in the musculoskeletal system are reduced.

Essentially, the back school curriculum is made up of three sections:

- education about the structure and functions of the back
- systematic following of the back school rules (see **Table 3.2**)
- active protection of the spine by means of physical therapy and sport; spine-stabilizing sports are suggested.

Back School and Orthopedic Pain Therapy

The following comments on the importance of postural and movement training programs in orthopedic pain therapy use back school as an example, but the same principles apply to joint classes, such as knee and shoulder classes.

The Aim of Back School

Back school is defined as "postural and behavioral training for the prevention of spinal damage." Note that the term "pain" is not mentioned in this definition. A reduction in perceived pain occurs later as a result of changed movement behavior.

Back School as a Primary Preventive Measure

Preventive back school aims to prevent pain occurring in the first place. It is designed for children, adolescents, and adults and takes place in many locations including kindergartens, primary and secondary schools, universities, community colleges, companies, and clubs.

It is not considered appropriate to mention terms such as "pain" or "symptoms" in the context of primary preventive back school. Repeated threats about the negative consequences of inappropriate behavior may prejudice the effectiveness of preventive programs in childhood and adolescent health education. From a didactic point of view, it is wiser to emphasize the positive effects of healthy behavior.

Back School as a Secondary Preventive Measure

Back school conducted as a secondary preventive measure aims to minimize symptoms and prevent them from worsening. These facilities are known as orthopedic back schools and are run by a team of physiotherapists and psychologists.

Participants in secondary preventive back schools are patients who are currently suffering from pain or have suffered from pain in the past. Our own research (Menzel 1996) and the meta-analysis by Maier-Riehle and Härter (1996) demonstrate that patients entering this type of back school already have chronic pain that has lasted 1–2.7 years. For this reason, one module of the orthopedic back school curriculum is dedicated to pain coping mechanisms (Nentwig et al. 1997). As suggested by Meichenbaum (1977), a simplified form of the gate control theory is explained so that participants can understand why they are recommended to participate in social and physical activities, even when they are in pain. In addition, a short form of Jacobson's progressive muscle relaxation technique is included in the pain coping module. However, the results of the meta-analysis by Maier-Riehle and Härter (1996; n = 18 studies), addressing the effectiveness of back school, demonstrated that the primary aim of changing behavior and education was achieved significantly more than the reduction of pain.

Back School and Pain Chronification

There are many reasons why classes addressing postural and movement behavior play an important role in orthopedic pain therapy.

NOTE

Back school is a prerequisite for orthopedic pain therapy.

Because of the pain they feel, patients cut down on many of their physical and social activities during the period of chronification. They avoid playing sports and participating in leisure activities, and go out less frequently. This reduction in social and physical activities leads to the pain intensifying and becoming constant. For this reason, motivation and encouragement to participate in these activities is an important component of pain therapy. It is essential for activities to be conducted in a spine- and joint-friendly manner, however, so that patients can follow the recommendations mentioned above. Patients therefore have first of all to be instructed in the appropriate movement patterns, and practice their execution. Confidence in knowing the rules of the back and joint class is a necessary prerequisite for pain therapy.

The psychological aims of orthopedic pain therapy include reducing the external locus of control, anxiety, and depression. In back school, participants are taught about the relationship between movement behavior and biomechanical changes. They gain a deeper insight into the anatomy, physiology, and pathology of their spine and joints. These factors increase their knowledge of their current disease and its potential development, and they can use this knowledge to better explain their symptoms and pain. They learn that sources of pain can be relieved with appropriate movement behavior. In this way, the external locus of control and feelings of helplessness can be reduced. An improved understanding of the causes of pain can also minimize participants' fears about the further progression of their disease. In addition, back and joint classes motivate participants to take part in various types of physical activity. These activities are partly conducted as "homework," with participants reporting back to the class afterward. This helps to counteract the occurrence of behaviors caused by depression.

Orthopedic pain therapy methods consist of multidisciplinary team work including, last but not least, psychology. Orthopedic patients are often unpleasantly surprised when they are introduced to a psychologist as part of their treatment. They experience their pain as coming from a concrete physical cause, and the introduction of a psychologist makes them worry that some unusual cause for their symptoms is suspected, or that they may be accused of malingering. Patients who have participated in orthopedic back school, endoprosthesis classes (Jerosch and Heisel 1996), or similar joint classes have already got to know the psychologist as a member of the team and have already learnt that psychologists can provide concrete help in dealing with pain. This significantly reduces the fear of starting psychological pain therapy.

4 Symptomatic Pain Therapy

Introduction

Symptomatic orthopedic pain therapy concentrates on nociception in bones, muscles, tendons, and joints after the pain stimulus has already acted on the body. This means that the nociceptive process has already started. Nociceptors have been irritated, and the pain signals have been transmitted to the brain via the afferent fibers and the spinal cord. Pain has already been perceived in the brain, and the motor and autonomic reactions in the periphery have been initiated. Symptomatic pain therapy acts on the different areas responsible for the transmission and perception of pain, and the reaction to it, with the emphasis varying according to the individual type of treatment (**Fig. 4.1**).

In symptomatic pain therapy, unlike causal pain therapy, the physician and patient expect an immediate response. Thus, fast-working analgesics, local injections, physical agents, and directly acting forms of electrotherapy form the main focus of treatment (**Table 4.1**).

Table 4.1 Symptomatic Treatment in the Spinal Region

Therapeutic local injection treatment (TLIT)
Medication
Thermotherapy
Physical therapy
Electrotherapy
Acupuncture

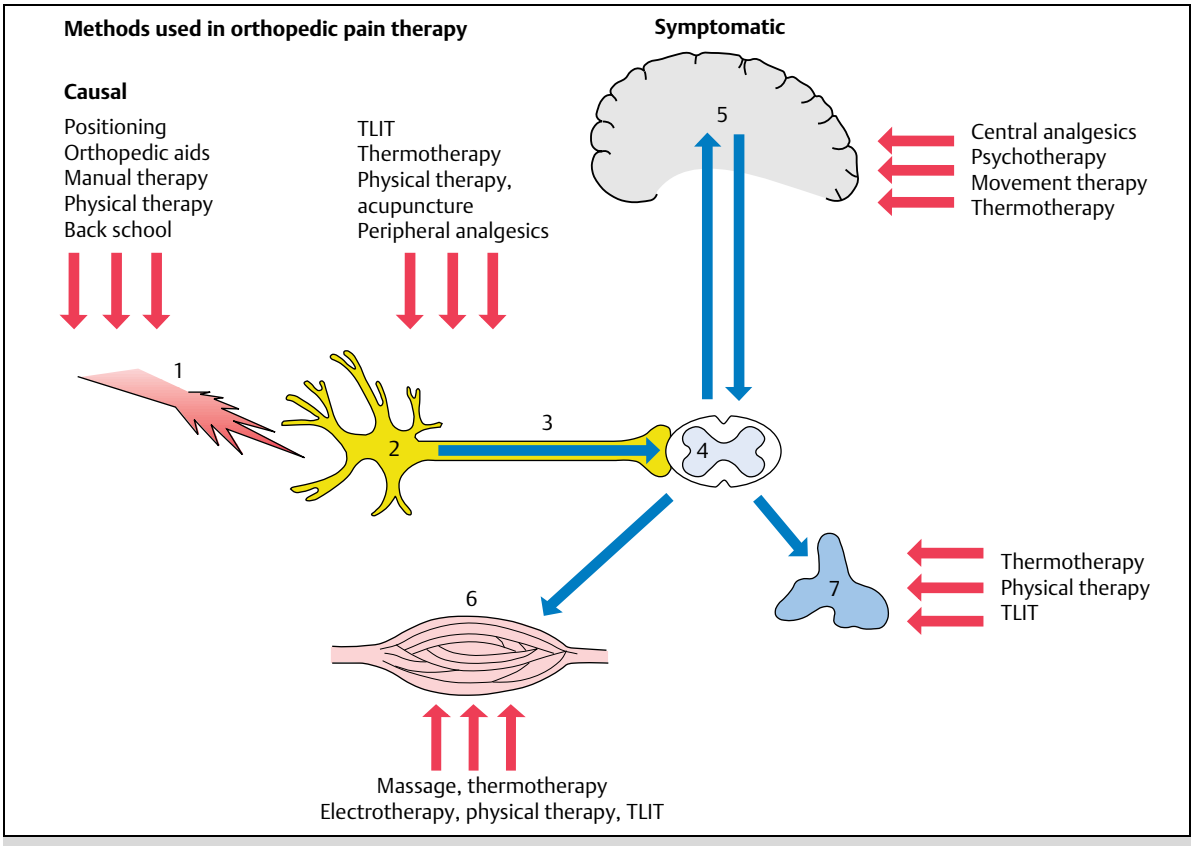


Fig. 4.1 Methods used in orthopedic pain therapy.

Thermotherapy

NOTE
All forms of warmth ease spinal pain.

In many cases patients apply soothing warmth before consulting a doctor. Warmth acts as a local analgesic, removing inflammatory mediators, relaxing muscles, and calming the autonomic nervous system. The cortex and the psyche perceive warmth as being pleasant (**Fig. 4.2**).

Forms of Application

Warmth can be applied in different ways. Heat can be transmitted either by placing the heat carrier in direct contact with the patient or indirectly by using radiant heat. The warmth from fango (volcanic mud used in spa treatments) and mud packs penetrates well into the skin. The simple application of dry heat, e. g., various forms of infrared treatment, has also proven effective in practice.

Heat pads, hot water bottles, and hot baths are immediate measures which are recommended for home treatment. The local thermal effects are, in decreasing order (Tilscher 1989):

- steam shower 52°
- hot shower 40–42°
- hot packs 40–42°
- mud packs 43–45°
- hay packs 43–45°
- hot air treatment (hot air box) 35°

The steam shower is the most intensive form of local heat application and is especially pleasant for patients suffering from chronic recurring cervical syndromes.

Contraindications for the local and generalized application of heat are thromboses and thrombophlebitis, cardiovascular disorders, dermatoses, acute inflammation, and florid infectious processes.

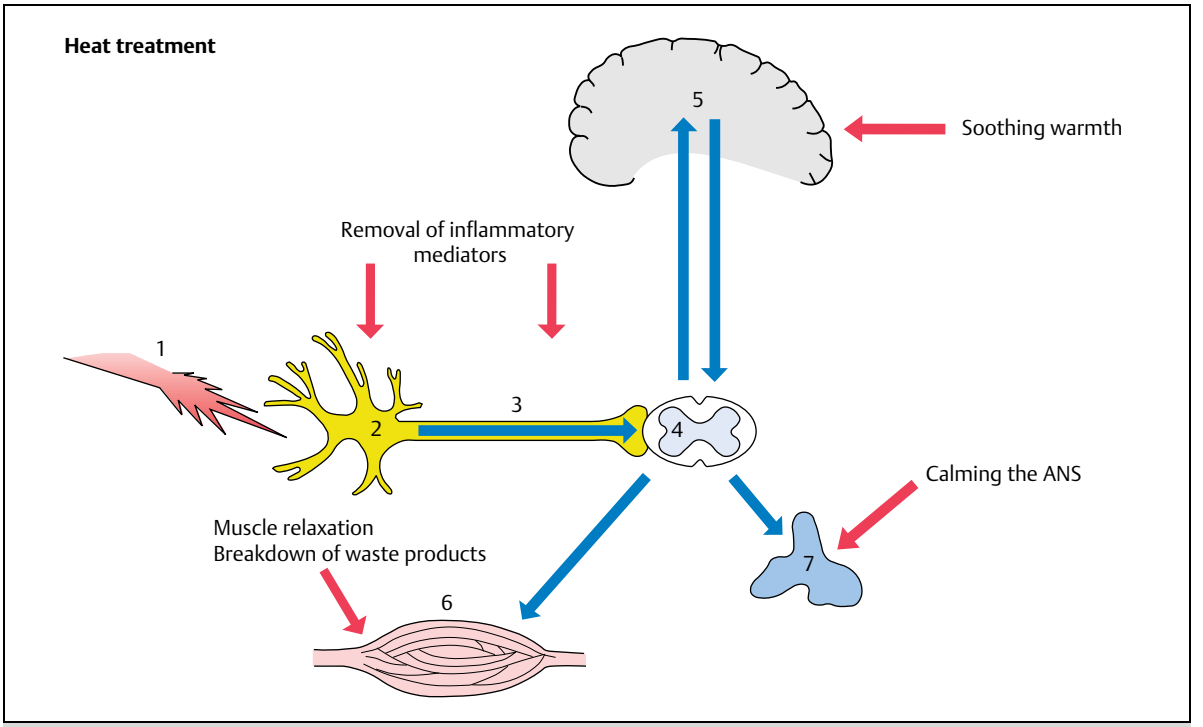


Fig. 4.2 The application of heat in the treatment of back pain.

Massage

NOTE

Massage is a special form of manual therapy used to treat painful spinal disorders.

The hands are used to massage skin, subcutaneous fatty tissue, muscles, and ligaments. A variety of techniques are applied during a massage:

- stroking and frictioning
- kneading and wringing
- cross-frictions.

Special forms of massage include reflex zone massage and connective tissue massage. The connective tissue massage technique involves a specific way of stroking with a fingertip (usually the third or fourth finger) with the hand, arm, and shoulder being held in a relaxed position. The direction of the strokes is segmentally orientated.

It is important that the patient is placed in a comfortable and relaxed position during all types of massage. This particularly applies to the affected body part. Massage acts positively on local nociception by removing inflammatory mediators. In addition, the vicious cycle of pain–muscle cramping–pain is disrupted at the muscular level (**Fig. 4.3**).

Contraindications to massage arise, as a rule, from acute pain, inflammation, skin changes, and nerve root compression syndromes.

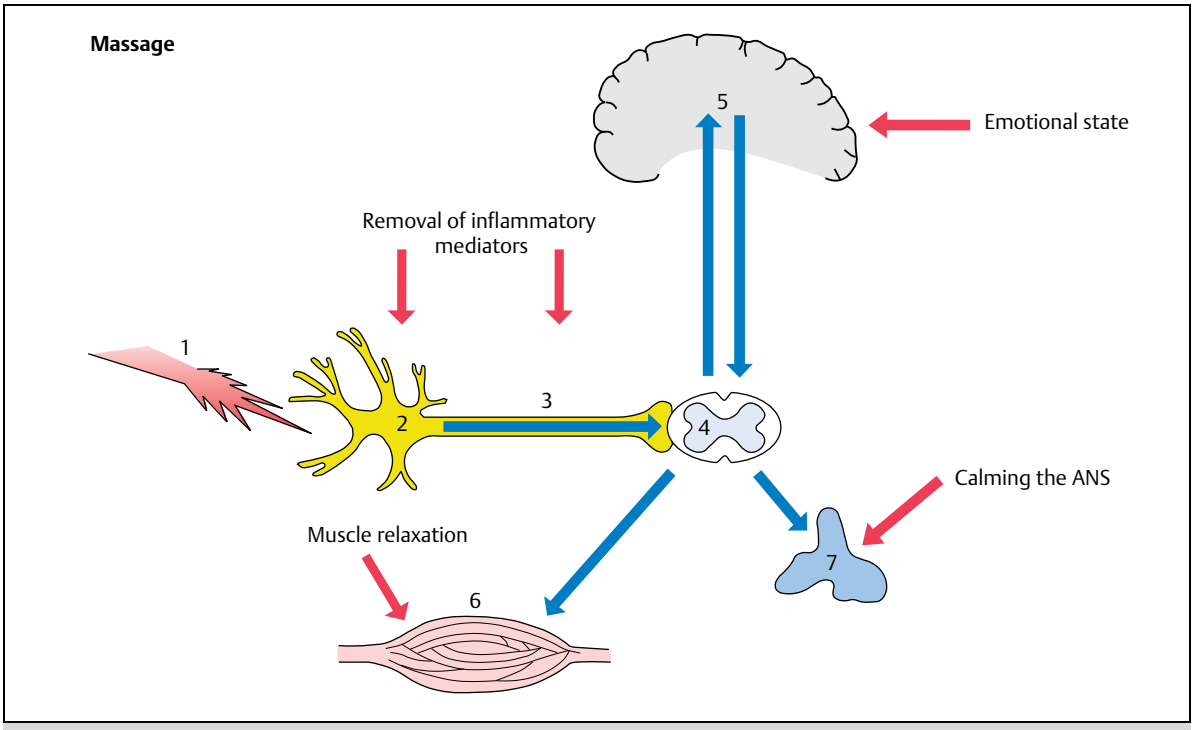


Fig. 4.3 Massage in spinal pain therapy.

Electrotherapy

In electrotherapy, electrical energy is used for the purpose of healing. Different types of currents vary in their physical and biological actions:

- **High-frequency currents** in the form of short waves, decimeter waves, and microwave therapy act by warming the depths of the tissue being treated. The oscillations are too fast to excite cells directly. The high-frequency currents that are applied therapeutically start at 20 000 Hz, increase to the diathermic ranges of ~3×10^6 Hz, and go up to the short-wave therapeutic range at ~5×10^7 Hz.
- **Low-frequency currents** (15–250 Hz) are used in galvanization, where direct current is applied. In this frequency range an increase in polarization or depolarization is found in the cells, with all transitional stages. A pain-relieving effect has been attributed to the constantly flowing direct current (e. g., in the form of a hydroelectric bath). Bernard's diadynamic currents are low-frequency currents with alternating frequency and amplitude, which also act to relieve pain. The electric currents are usually applied bipolarly, i. e., using two electrodes.
- **Mid-frequency currents** are sinusoidal alternating currents with frequencies between 1 and 1000 Hz. The principle of mid-frequency or interferential current treatment involves the production of biologically effective frequency ranges within the organism itself. In interferential treatment, two biologically nonirritant mid-frequency currents (e. g., 4000 Hz) are applied to the body using two electrodes. The frequency of the currents differs by up to 100 Hz. Their superposition results in the development of an amplitude- and frequency-modulated current with lower effective frequency, i. e., a frequency that is biologically effective within the body. The advantages of interferential treatment are twofold: the mid-frequency stimulating current effectively overcomes the pain-sensitive skin and outer tissue layers, while the low-frequency currents (between 0 and 100 Hz) are first generated in the deeper tissue layers and function there as a form of **pain therapy.** In this way, electrical currents that cannot be applied directly from external sources at this frequency and intensity are developed at the desired location within the body. The direct action of the low-frequency current in the deeper tissue affects the autonomic nervous system and improves blood flow.

High-frequency, low-frequency, and interferential therapy act similarly on nociceptors, afferent fibers, muscles, and the autonomic nervous system (**Figs. 4.4, 4.5**).

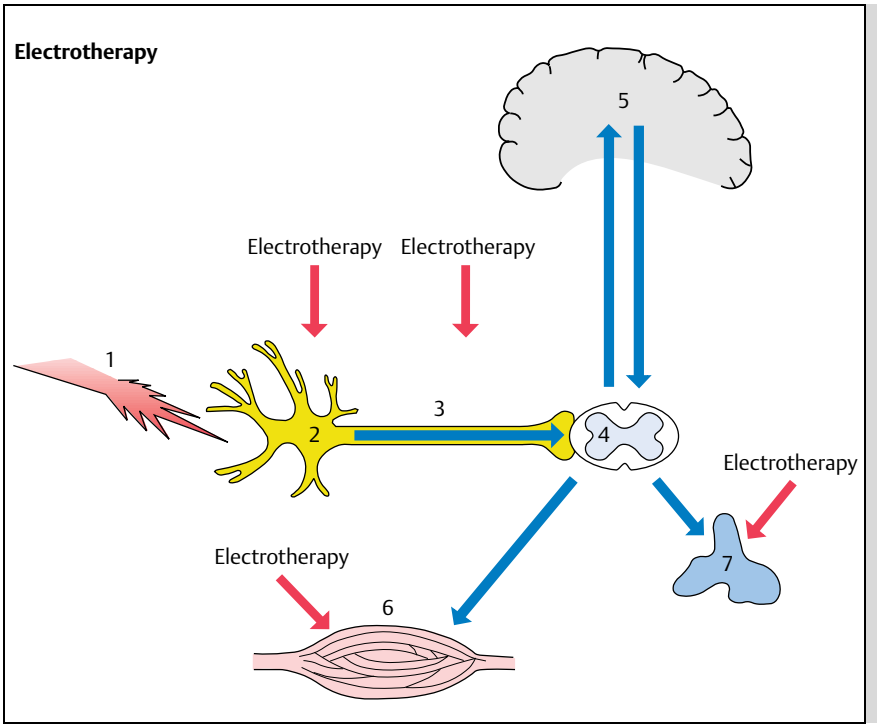


Fig. 4.4 Electrotherapy in spinal pain therapy.

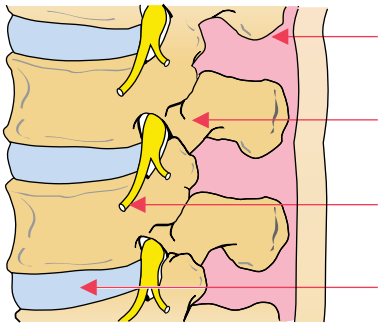


Fig. 4.5 The differentiated indications for electrotherapy on the spine (according to Niethard and Pfeil 2003).

Acupuncture

The word **acupuncture** comes from the Latin *acus,* "needle," and *pungere,* "to prick." Both traditional and classical Chinese medicine use a technique known as *zhēn jiǔ* (= insertion and burning, referring to acupuncture together with moxibustion). Fine needles are inserted into specific points on the body. The Chinese term for these acupuncture points is *shu xue*, where *shu* means transporting or conducting, and *xue* means cavity or hole.

NOTE

Acupuncture is the practice of inserting needles into specific areas of the skin. Among other effects, it activates the body's own pain inhibition.

According to Heine (1987), ~80% of the traditional acupuncture points have an anatomical counterpart. Morphologically they can be described as perforations of the superficial body fascia tissue by a bundle of blood vessels and nerves.

The analgesic effect of acupuncture is probably due to the release of endorphins, which ease pain. According to Pomeranz (1981), the peripheral pain stimulus activates the analgesic action. This gives rise to a mechanism that acts in three stages. The pain caused by the needle prick acts as a noxious stimulus (1 in **Fig. 4.6**), stimulating the nociceptor (2). The pain signals are then further transmitted to the posterior horn of the spinal cord (4) via the afferent fibers (3). It is here that the pain signals are transferred to a second neuron, which in turn transmits the pain signals up to the thalamus and finally to the cortex (5), the place where pain is localized. The body's own opioid peptides (endorphins) inhibit the transmission of nociceptive information, acting on the synapses of the nociceptive system in the spinal cord (4) and the brain (5). Endogenous opioids are released as inhibitory transmitters from the neurons. These neurons can be thought of as part of the antinociceptive system that is activated by acupuncture (Stux et al. 2003).

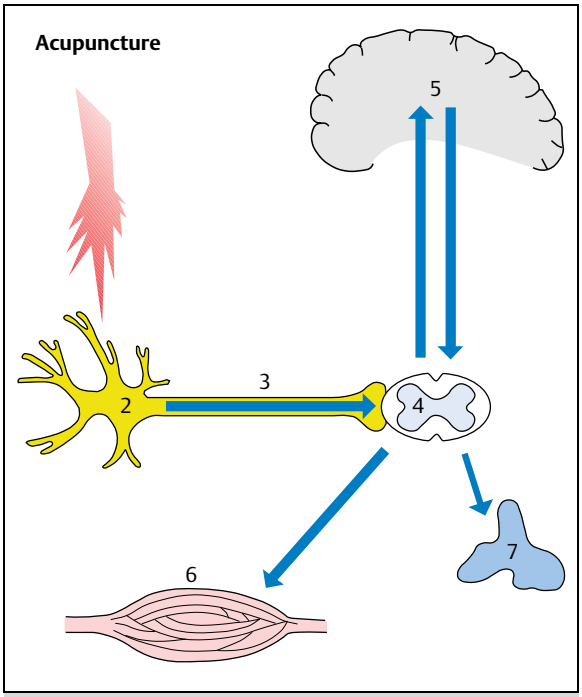


Fig. 4.6 Acupuncture and nociception. The pinprick from acupuncture acts as a noxious stimulus (1), stimulating the nociceptor (2). The stimulus is transmitted to the spinal cord (4) via the afferent fibers (3) and from there via the spinothalamic tract to the brainstem (5). Pain is not perceived. Pain-inhibiting mechanisms are sent to the periphery via the descending pathways and act as an analgesic.

The classic body acupuncture can be used to treat all forms of chronic pain, acting an adjuvant form of treatment within orthopedic pain therapy. The treatment should preferably be performed in a stress-relieving position, i. e., with the spine relaxed (**Fig. 4.7**).

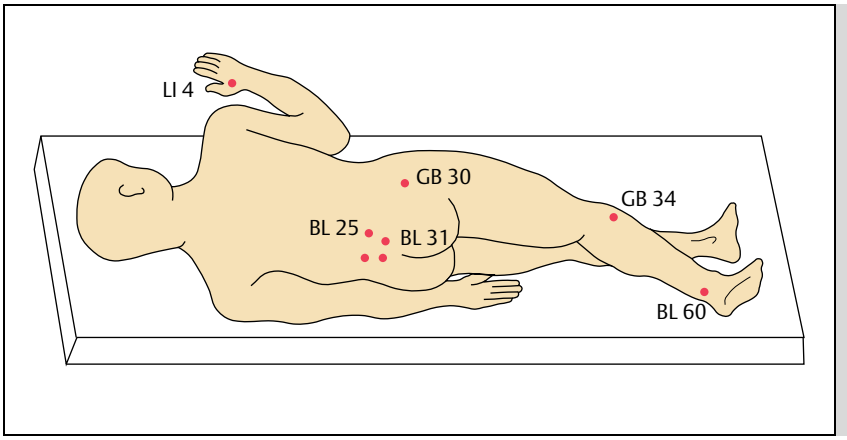


Fig. 4.7 Acupuncture program, e. g., for sciatica. The patient lies relaxed on one side with hips and knees bent. The painful side is uppermost. The needles are inserted one after the other, starting with the foot. The needles are inserted between 2 mm and 15 mm deep and are removed after 15 minutes. The acupuncture points are essentially found in the areas of pain or radiation (acupuncture points according to Stux et al. 1993).

We have assessed pain patients during a comparative study on the effectiveness of acupuncture within the scope of pain therapy. In this study acupuncture points were not individually chosen, but were rather based on standardized points for needle insertion (Grifka and Schleuß 1995). The study established that acupuncture using the traditional acupuncture points is significantly more effective than placebo acupuncture, where points were randomly chosen. The reported values for the total amount of pain and the highest level of pain were significantly lower for patients treated with traditional acupuncture over 14 treatment sessions than for patients treated with placebo acupuncture (Grifka and Schleuß 1995). However, the attitude of individual patients to the effectiveness of acupuncture plays an important role (Grabow 1992). A meta-analysis demonstrated that 75 out of 88 studies showed a positive result when acupuncture was used in pain therapy (Molsberger and Böwing 1997).

At least 10 acupuncture treatment sessions of at least 15 minutes each are required for effective treatment.

Therapeutic Local Injection Treatment

NOTE

It is possible to treat the primary disorder by injecting fluids with anesthetic, anti-inflammatory, and anti-edemic properties directly into the nociceptive source in the spine (**Fig. 4.8**). This avoids loading the entire body with more medication than necessary.

The patient's medical history and the results of manual medicine examinations provide important clues to the choice of site for the local therapeutic injection (see Chapter 2, "Clinical Examination"). Further guidance can be obtained from diagnostic local anesthesia or local pain provocation using saline solutions or contrast agents (see Chapter 2, "Trial Measures for the Diagnosis of Pain").

Local anesthetics, steroids, or a combination of the two are used for the therapeutic local injection, depending on the aim of the treatment. Pure saline solution is also sometimes used: the hypertonic solution has an osmotic effect on the edemic tissue and dilutes the accumulated inflammatory mediators.

Therapeutic local anesthesia (TLA) is a fundamental part of the therapeutic local injection treatment (TLIT). A few milliliters of dilute (0.5–1.5 %) local anesthetic solution are all that is needed to switch off sensitized nociceptors and nerve fibers that have developed into nociceptors. This results in the following effects:

- pain reduction
- decreased nerve excitability
- increased local blood flow.

Nociceptors and afferent fibers are reversibly switched off after local anesthetics have infiltrated the tissue. This abolishes the excitability of pain transmission, sensitive end organs, and the ability of sensitive sections of nerve fibers to transmit signals, and does so in a reversible manner at a local level. The effectiveness of local anesthesia decreases as nerve fiber diameter increases. For this reason, sensitive nerves are initially blocked and motor nerve fibers are blocked when high doses are injected. Therapeutic local anesthesia is directed toward the sensitive nerve fibers. Local anesthetics reduce the permeability of the membrane to cations, especially sodium ions. This results in diminished membrane permeability with reduced levels of excitability.

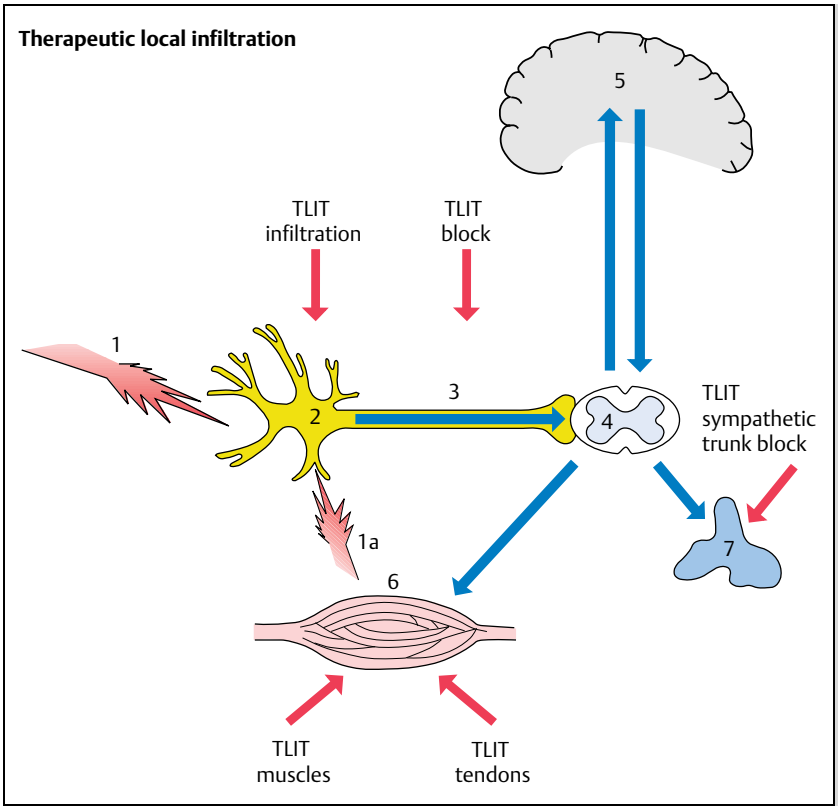


Fig. 4.8 The influence of TLIT on the nociception in the musculoskeletal system. The nociceptors and afferent fibers are switched off using local infiltration or nerve blocks (2 and 3). The nociception–muscle tension–adaptive posture cycle (2, 3, 6, 1a) is disrupted by the use of therapeutic local infiltration into tendon attachments and muscle infiltration. The autonomic reaction (7) is switched off using sympathetic chain blocks.

NOTE

The use of high concentrations with complete anesthesia and paralysis is not necessary during the local infiltration treatment. **The aim is to decrease excitability and increase irritation thresholds.**

Neurophysiologically based TLA measures break the link between muscle tension and the excitation of nociceptors (Zimmermann 1993). A nociceptor or nerve block results in a reduction in pain and nerve excitability and an increase in local blood flow for a period of 3–8 hours, depending on how long the applied local anesthetic works effectively. Experience shows that the pain-relieving effect is maintained longer than would be expected from the local anesthetic's duration of action. This is especially the case with repeated administration. **The state of reduced excitability continues, so that it is possible to obtain a permanent effect with a series of 8–12 infiltrations on consecutive days.**

Infiltrating a local anesthetic multiple times into the area of nociception and the outgoing afferent fibers results in a desensitization of overactive neural elements. The frequency and intensity of the transmitted excitatory impulses that are required for pain perception and motor or autonomic reactions decline.

NOTE

Repeated administration of TLA prevents the development of pain chronifaction (**Fig. 4.9**).

If chronification has already occured, the use of repeated TLA disrupts the vicious circle of adaptive posture–nerve irritation–increased muscle tension and pain at the neural level. The desensitization of nociceptors and afferent fibers, with an increase in irritation thresholds, leads to the same mechanical stimuli causing less pain. In this phase causal pain therapy has to be implemented by relieving positioning, exercises, etc. In chronic spinal pain syndromes, the repeated use of TLA in the area of nociception and afferent fibers results in a reduction in pain perception and pain processing (Zieglgänsberger 1986).

Rydevik's (1990) and Olmarker's (1993) groups studied the reactive inflammatory changes in nerves and nerve roots (e. g., due to prolapsed intervertebral disk tissue). They demonstrated that a defined chronic compression evokes an inflammatory and edematous change in the nerve root. This change can be largely prevented by the use of lidocaine injections (Yabuki et al. 1996).

Most local anesthetics also act as vasodilators, so blood flow increases markedly in the infiltrated area. However, this also means that injected medication is more rapidly removed by the circulatory system. In most cases, however, there is no indication for the addition of vasocon-

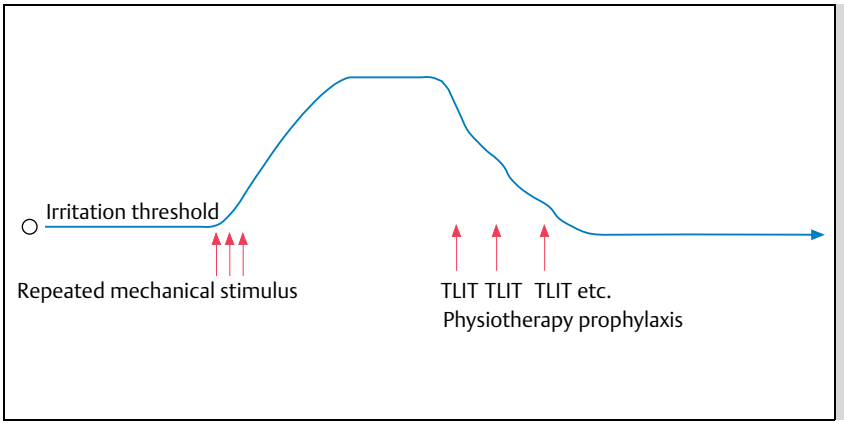


Fig. 4.9 The reduction in the excitability of nociceptors and afferent fibers by repeated use (8–12 times) of therapeutic local anesthesia. The irritation thresholds of nociceptors and afferent fibers, which have been raised by the repeated pain stimuli, revert to their normal level. The additional use of electrotherapy with positioning, physical therapy, and heat treatment further strengthens this effect.

strictors to the local injection treatment used for spinal symptoms. Notable specific side effects of the administration of local anesthetics are cardiovascular complications when the blood levels are too high, and allergic reactions. These complications are rare, however.

A maximum of 10 mL of 0.5–1 % local anesthetic is used at each injection treatment session in order to avoid elevated blood levels. Intravascular application is avoided by constant aspiration.

Reliable Medications for Therapeutic Local Injection Treatment

- **Lidocaine** (0.5 %) is a fast and long-acting local anesthetic.
- **Bupivacaine** (0.25 %) is lipophilic and is preferably used as a long-term anesthetic. When used at concentrations of up to 0.25 %, it results in long-lasting analgesia with motor activity remaining largely unaffected.
- **Ropivacaine** (2 mg/mL) was the first local anesthetic tested where clear, unambiguous results were obtained. Its blocking behavior demonstrates an advantageous weighting of sensory to motor effect.

Steroids are also infiltrated initially and during further treatment with local injections, as part of orthopedic pain therapy. The treatment focuses on the concomitant inflammatory reaction in the nociceptors and the area surrounding the afferent fibers. **Steroids neutralize the pain-evoking prostaglandins and leukotrienes** (Wehling 1993). For this reason, they have a local **analgesic** effect in addition to their **anti-inflammatory** action. The use of steroids with a high receptor affinity, such as triamcinolone, is preferable. A sufficiently high concentration of active ingredients in the immediate proximity of irritated structures is needed to ensure an effective pharmacodynamic interaction of steroids with the circumscribed inflammatory processes. The use of general medication such as orally administered steroids is therefore not a part of orthopedic pain therapy and is used only in exceptional cases. The concentration of steroid at the source of pain should be maintained for several weeks. At the same time, the systemic perfusion of glucocorticoids should be kept to a minimum in order to limit the pharmacodynamic loading on the entire organism. These guidelines can best be followed by using glucocorticoid depot preparations in the form of crystal suspensions. We therefore mainly use triamcinolone diacetate and triamcinolone acetonite when treating acute and chronic radiculopathies. Our research (Barth et al. 1990) has demonstrated that local administration of 5–10 mg of these steroids can saturate all steroid receptors in the surrounding tissues. Significant side effects (e. g., a sustained suppression of the body's own cortisol production) are not to be expected when the steroid is administered in this form 1–3 times as part of a treatment cycle for a pain syndrome. Allergic reactions to carrier substances in steroids and local anesthetics are, however, to be anticipated when using all types of medication (see Chapter 10).

Therapeutic local injections are administered to painful muscle and tendon insertions as well as to different locations in the vertebral motor segments. The indications and techniques for individual injections can be found in the atlas section of this book.

Interleukin-1 Receptor Antagonist Protein (IL-1Ra)

As early as the 1920 s, in connection with research into tuberculosis, it was already being speculated that special proteins act as messengers in "cell communication" (Zinsser and Tamiya 1926). Since then, a succession of new proteins has been characterized and an increasingly complex network of messengers has been discovered, initially with inconsistent nomenclature. In 1991 all mediators were for the first time grouped together under the term **cytokines**, and a systematic naming system was introduced (Klein 1991).

Interleukin 1 (IL-1) was first described in 1940, under the name "endogenous pyrogen." It was subsequently found that this protein can be detected in all cells of the body, and that IL-1 receptors exist (Dower et al. 1984). Later, a naturally occurring antagonist was discovered (Liao et al. 1984) and was named anti-interleukin 1 or, more correctly, the interleukin 1 receptor antagonist protein, abbreviated to IRAP or IL-1Ra.

When the biological actions of IL-1 were studied, it was found to participate in the genesis and maintenance of acute and chronic inflammation as well as in the destruction of tissue. The nerve root, along with the articular cartilage, is an important target tissue in this context. More precise studies and experiments were able to demonstrate that a disproportionate ratio between agonist and antagonist seemed to govern certain disorders (Lennard 1995). Conditions where the proportion of IL-1 to IL-1Ra plays a central role include osteoarthrosis; intervertebral disk pathologies and nerve irritation syndromes that clinically present as lumbago or as lumboischialgia; and rheumatoid arthritis, especially the destruction of cartilage.

IL-1Ra is the only naturally occurring antagonist so far discovered within the cytokine family (Arend et al. 1989). As it acts by binding to the IL-1 receptor, the competitive binding of receptors seems to play a decisive role in pathology. An excess of IL-1Ra has to be present to repress IL-1 enough to antagonize its biological actions (Arend et al. 1990, Seckinger et al. 1990). This is the starting point when looking at the direction treatment should take. In certain disorders, such as osteoarthrosis and rheumatoid arthritis, there is a surplus of IL-1 relative to the levels of IL-1Ra. For this reason efforts are being made to administer IL-1 receptor antagonists exogenously in order to neutralize the action of IL-1.

The Role of IL-1 in Relation to Neural Structures

By the 1990 s it was already known that that cytokines, especially IL-1 acting as the primary mediator, could be responsible for neurological deficits and the development of pain. In the case of lumbago or lumboischialgia, mediators, primarily IL-1, are produced in the small vertebral joints or the intervertebral disk. These mediators are able to reach the area immediately surrounding the nerve roots and initiate pathological processes in the form of nerve inflammation and deterioration of function (Wehling 1991, Wehling et al. 1996). This knowledge resulted in the use of IL-1 receptor antagonists in the causal treatment of these symptoms.

Causal Therapy Using the IL-1Ra/ Orthokine/EOT Technique

The Orthokine technique and the more recent forms of the so-called EOT technique have made available for the first time an autologous IL-1Ra that acts causally in the breakdown process of joint wear or the inflammatory process at the nerve root.

It is known that IL-1 and the naturally occurring IL-1Ra can be found in all cells of the body, including certain blood cells and in particular the monocytes (Lennard 1995, Dinarello 1991). It has also been proven that some substances, superficial structures, and materials activate the production and release of IL-1Ra (Arend 1991 a, b). The Orthokine technique takes advantage of this fact by extracting venous blood from the patient with a specially developed syringe. The Orthokine syringe or EOT syringe contains glass beads which are made in such a way that their surface structure activates the monocytes to produce more IL-1Ra. The IL-1Ra-enriched protein concentrate can be separated following a specific incubation period at 37 °C, and extracted from the syringe without the addition of additives. The direct administration of active ingredient into the affected nerve root allows the ingredients to reach the affected area. The local enrichment of the autologous IL-1Ra at the inflamed nerve root causes an anti-inflammatory and anti-edemic effect by means of the occupation of receptors, thus switching off the action of IL-1.

The Orthokine method involves the physician taking approximately 60 mL of blood using the Orthokine syringe. The syringe is placed in an incubator and sent to the sterile laboratory at the manufacturing company Orthogen AG. The blood is serologically examined there, and the IL-1Ra-enriched serum is extracted and frozen. The treating physician receives at least six ampules of serum within about a week, following clearance from the laboratory.

The more recent EOT method is based on the same principles. In this method, however, treating physicians can manufacture the IL-1Ra-enriched serum themselves in a simple procedure using the EOT blood collection system. The special 5 mL Luerlock syringes used to take blood produce enough serum for injection (~2–4 mL). Physicians can therefore decide themselves how many ampules are required, depending on the presenting disorder. The serum is available for injection after 6–9 hours of incubation; the waiting period of several days is no longer necessary. Serological testing for HIV, hepatitis B and C, and syphilis

Figure 4.10 Taking blood samples using the EOT Syringe System under aseptic conditions. Each patient requires 4–6 tubes, depending on the intended use.

Fig. 4.11 The tubes (labeled with the patient's data) are incubated for 6–9 hours at 37°.

Fig. 4.12 The blood-filled tubes are centrifuged at 5000 rpm for 10 minutes to separate out the serum. Tubes for only one patient are placed in the centrifuge at any one time.

should be carried out before blood is collected (**Figs. 4.10–4.15**).

Treating the Nerve Root and Intervertebral Disk using Autologous IL-1Ra

Orthokine has been used for several years in the treatment of intervertebral disk pathologies and nerve root irritation. Nerve irritation syndromes can be effectively treated with the specific administration of medication at the nerve root in the form of a nerve root block and epidural administration. Intradiscal administration, where indicated, is also appropriate. In addition, Orthokine can be successfully injected into arthrotically altered facet joints. Further statements are expected following the completion of clinical studies that are currently in progress. Orthokine is approved in the EU and in Australia.

Recombinant IL-1Ra

Human IL-1Ra has been successfully cloned. Recombinant IL-1Ra has been successfully applied experimentally and in the treatment of rheumatism, where it has been used as a systemic treatment by means of subcutaneous injection (Carter et al. 1990, Arend et al. 1991 a, b, Smith and Arnett 1991, Campion et al. 1996, Bresnihan et al. 1998 a, b).

Recombinant IL-1Ra has, however, several disadvantages in comparison to the autologous substance. The glycosylation of the protein varies between individuals, and it appears that higher surplus concentrations of the recombinant version are needed to repress the IL-1 at the receptor. In addition, a reaction occurring at the point of needle insertion has frequently been observed (Antin et al. 1994, Bresnihan et al. 1998). Finally, as the human DNA is combined with additives, the possibility of potential allergic reactions should be considered.

Clinical Results

Many clinical studies have established the effectiveness of various active ingredients administered in the epidural perineural injection treatment of lumbar nerve root compression syndromes (Carette et al. 1997, Krämer et al. 1997 a). So far, only one clinical study has assessed the effectiveness of IL-1Ra-enriched serum compared with

triamcinolone in this type of treatment (Becker et al. 2007). In this prospective randomized double-blind study of 90 patients, 5 mg and 10 mg of autologous IL-1Ra were compared with triamcinolone under identical test conditions. Measurements were made at six follow-up sessions, at 6-monthly intervals. These patients suffered from MRI-verified lumbar nerve root compression symptoms where surgical intervention was not indicated. Further therapy, except oral PRN medication, was not permitted during the follow-up period. The treatment effect was recorded using a standardized clinical examination and questionnaires (SF-36, SES, VAS, Oswestry Score, pain diary). The effectiveness of therapy was assessed over the entire examination period by means of general linear models. All groups demonstrated a significant improvement in their symptoms. The assessment of the differences between the groups over a period of time showed, for example, a significant difference in pain levels ($p < 0.05$) after 3 months when measured with the VAS. The IL-1Ra group experienced a lasting reduction in pain while the triamcinolone group tended toward a further increase in pain.

Conclusion

Serum enriched with IL-1Ra (e.g., Orthokine) offers an alternative form of therapy for the treatment of nerve root compression syndromes. When the diagnosis has been correctly made, a significant improvement in symptoms can be achieved over an average period of 3–6 months. It has additionally been proven that the epidural perineural application involves minimal risk and that systemic side effects do not occur.

Fig. 4.13 The tubes and puncturable membrane are sprayed with disinfectant, which should be left to work for at least 1 minute. The tubes (from one patient at a time) are then placed on a previously prepared work surface.

Fig. 4.14 The serum is carefully extracted using a 5 mL Luerlock syringe. Air is pulled into the syringe to make extraction easier and then pushed slowly into the tube after the rubber membrane has been penetrated.

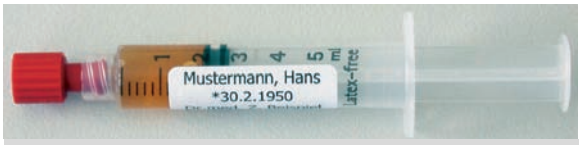


Fig. 4.15 The syringe is sealed with a suitable closure. Patient data are attached to every syringe. The syringes for each patient are stored in a separate box and frozen at –20 °C until required.

Multimodal Medication Concomitant Therapy

Introduction

An understanding of nociception and its regulatory systems is important in planning and conducting multimodal medication concomitant therapy for the treatment of spinal disorders (see Chapter 1, "Nociception and the Evolution of Chronic Pain"). From the triggering of the stimulus in the periphery to the final perception of pain in the cortex, pain can be modulated and repressed by medication that acts specifically on the individual systems.

The **cyclooxygenase system** is important in nociception, and cyclooxygenase inhibitors are administered predominantly in the periphery. In contrast, **opioids** bind to specific receptors in the CNS. Opioids can also act on peripheral opioid receptors that have been either newly generated in tissue altered by inflammation or on peripheral opioid receptors that have been placed in a functional standby mode. It therefore makes sense to administer opioids as a regional analgesic, e. g., intra-articularly.

The transmission of a nociceptive signals originating in the periphery is linked to the function of Aδ- and C-fibers. Signal transmission takes place via polarization and rapid sodium influx into the cells. Depending on the dose administered, **local anesthetics** prevent this transmission of signals to a certain extent, up to and including a complete nerve block. The transmission of nociceptive signals to the first interneuron at the spinal cord level is associated with many regulatory processes. Cell excitation is transmitted by the release of excitatory transmitters such as substance P, CGRP, glutamate, or neurokinin. A variety of filtering and modulating processes act on the pain impulse when it is transmitted from the first afferent neuron to the second neuron. Descending mechanisms for pain inhibition and inhibiting neurotransmitters such as endorphin and enkephalin play an important role here. Pain stimuli that are especially strong or long-lasting can cause permanent cellular changes at the spinal cord level ("neuroplasticity") so that a permanent increase in perceived pain can take place in certain cases. These sensitization processes occur especially when the so-called NMDA receptor channels are opened as a result of the repeated excitation of spinal cord neurons and an increase in calcium influx takes place. **NMDA receptor antagonists,** such as ketamine, influence spinal sensitization even in small doses.

Opioids bind to μ- or δ-receptors at the spinal cord level, causing a reduction in calcium influx and an increase in the dose-dependent polarization wave in nociceptive interneurons. Inhibition of nociception in the spinal cord also takes place as a result of the action of **α-2 agonists** (clonidine), **GABA-B agonists,** and **5-HT agonists** (serotonin).

NOTE

It therefore makes sense in the clinical environment to combine different medications (e. g., cyclooxygenase inhibitors, opioids, local anesthetics, α-2 agonists, NMDA receptor antagonists) as part of a multimodal medication concomitant therapy. In addition to this list are the **adjuvant analgesics;** these are not pain medication as such, but because of their special characteristics they are able to reduce the amount of analgesics required and thus minimize the sometimes considerable side effects.

Pain Measurement and Documentation

To record the pathophysiological cause of pain, it is essential to take an accurate individual pain history. This includes pain intensity when resting and when weight-bearing, the location of pain and area of spread, the quality of pain, the behavior of symptoms over a 24 hour period, and the subjective assessment of pain-aggravating and pain-relieving activities. A knowledge of all these factors is essential when creating a multimodal medication therapy plan, to do the individual circumstances and characteristics justice.

First, it has to be decided if the nociceptive pain has a somatic or visceral genesis, or if the pain is neuropathic (see Chapter 1, "Nociception and the Evolution of Chronic Pain"). Neuropathic pain can be found in the peripheral nerve-conducting channels or in the CNS. In so-called psychogenic pain, in the form of a somatoform pain disorder, the pain is influenced by serious psychosocial factors.

The information acquired from the medical history regarding the quality of pain (e. g., clear, burning, stabbing, shooting, deep, dull, or gnawing) gives clues as to which pharmaceutical adjuvant analgesics are the most suitable (e. g., antidepressants, anticonvulsants, glucocorticoids). The behavior of pain symptoms over a 24 hour period governs, for example, whether a higher dose is needed during the day or at night. The activities that subjectively influence the pain must not be neglected. Most patients tend to find their own way of dealing with the pain over the course of the illness. It is imperative to question patients about the type of coping strategies they use and to support these strategies when appropriate. Physical measures, such as heat, cold, or certain positions or postures, can play an important role in the reduction of pain medication.

Thorough pain measurements, their documentation, and the precise taking of the pain history provide a further basis for efficient pain therapy. The estimation of pain intensity is always subjective, except with young children

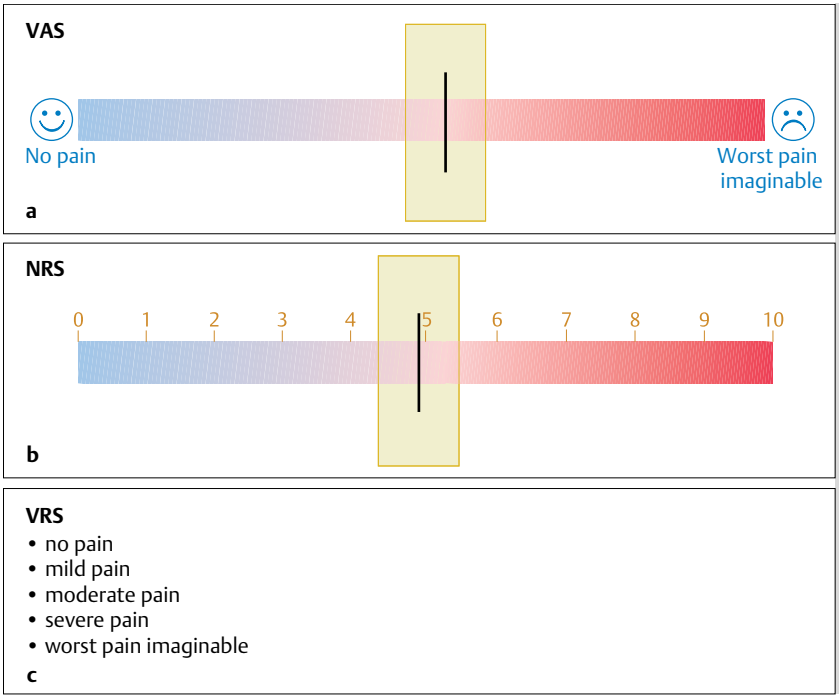


Fig. 4.16 a–c Validated pain measurement instruments:
a Visual analogue scale (VAS)
b Numeric rating scale (NRS)
c Verbal rating scale (VRS)

or demented or nonresponsive patients where it relies on third-party observation. A variety of validated instruments are available for reporting pain intensity. The **visual analogue scale (VAS)** measures the intensity of pain on a straight line labeled "no pain" at one end and "worst pain imaginable" at the other. The patient marks the line at the point best representing their pain intensity and this is then read off a scale from 0 to 100 on the other side of the paper (**Fig. 4.16a**). The **numeric rating scale (NRS)** involves patients numerically rating their pain, e. g., 0 = no pain, 5 = moderate pain, and 10 = worst pain imaginable (**Fig. 4.16b**). Some patients, especially elderly people, find the **verbal rating scale (VRS)** (**Fig. 4.16c**) easier to use. The patient chooses between verbal items such as:

- no pain
- mild pain
- moderate pain
- severe pain
- worst pain imaginable.

NOTE

In summary: multimodal medication pain therapy is based on the specific use of nonopioids, opioids, local anesthetics, and adjuvant medication in an individually adapted dosage on the one hand, and the regular monitoring of therapy with the appropriate documentation on the other hand.

WHO Analgesic Ladder

In principle, every type of pharmaceutical pain therapy has its own level on the WHO analgesic ladder, whether the pain is acute or chronic (**Fig. 4.17**).

Nonopioids in combination with adjuvant medication are used at level I. Level II involves the combination of mild opioids with nonopioids and adjuvant medication. For severe pain (level III), the administration of strong opioids together with nonopioids and adjuvant medication is required.

NOTE

For the treatment of chronic pain the WHO analgesic ladder additionally recommends that medication should, if possible, be administered noninvasively, in doses tailored to the individual patient, and at specific times rather than on demand (PRN).

Nonopioids

In most cases, the action of nonopioid analgesics is based on the inhibition of cyclooxygenase and thus of prostaglandins. The prostaglandins dilate capillaries and so amplify the reaction to chemical, mechanical, and thermal stimuli, facilitating inflammation and causing hyperalgesia. Prostaglandins also have important physiological functions, such as the regulation of blood flow in the kidneys,

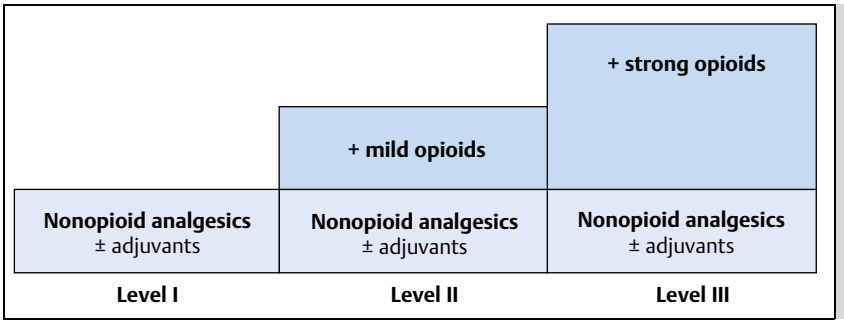


Fig. 4.17 WHO analgesic ladder.

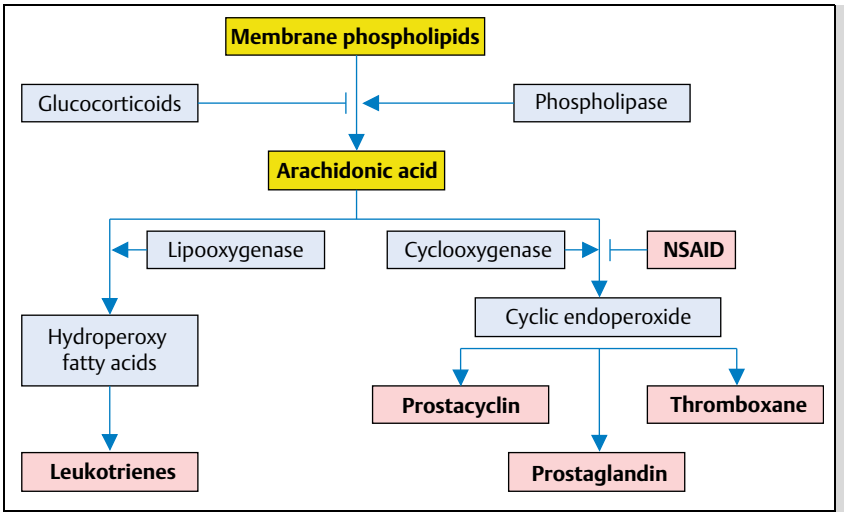


Fig. 4.18 The mechanisms of action found in cyclooxygenase inhibition.

Table 4.2 Specific Actions of Nonopioid Subgroups

Effect	*Acids (NSAIDs, coxibs)*	*Aniline derivative*	*Pyrazolone derivative*
Analgesic	+++	++	+++
Antipyretic	+	++	+++
Anti-inflammatory	+++		
Spasmolytic		++	

the tubular transport of sodium and water, and the formation of gastric mucus.

The various classes of nonopiod analgesics are categorized below. Metamizole and paracetamol act partially at a central level (**Tables 4.2, 4.3**).

- Acidic antipyretic analgesics:
  - salicylates (e. g., aspirin)
  - nonsteroidal anti-inflammatory medications (NSAIDs)
  - coxibs (selective COX-2 inhibitors).
- Nonacidic antipyretic analgesics:
  - aniline derivatives (e. g., paracetamol)
  - pyrazolone derivatives (e. g., metamizole).
- Nonopioid analgesics without antipyretic or anti-inflammatory actions:
  - flupirtine
  - nefopam.

Acidic Antipyretic Nonopioids

NSAIDs and coxibs act not only as antipyretics and analgesics, but also as anti-inflammatories, by enriching the acidic environment. Their use is indicated only in acute inflammatory processes because of their serious side effects.

NOTE

The incidence of life-threatening complications significantly increases in older people, and in patients with renal function disorders or cardiovascular risk factors.

Traumatic or inflammatory tissue damage results in the release of inflammatory mediators such as prostaglandin, histamine, and bradykinin. Each substance is inactive on its own, but when combined they cause strong pain reactions (Waldvogel 2001). It is at this point that NSAIDs act (**Fig. 4.18**), and by the end of the 1970 s it was recognized that aspirin and other similar substances inhibit the synthesis of prostaglandin.

Table 4.3 Overview of the Most Clinically Important Nonopioids

Non-proprietary name	***Paracetamol***	***Metamizole***
Preparations	Tablet 500 mg Suppository 125/250/500/1000 mg Syrup 1 mL = 200 mg Prefabricated infusion solution 1000 mg	Drop solution 1 mL = 500 mg Tablet 50 mg Suppository 300/1000 mg Ampules 1 g/2.5 g
Single dose	500–1000 mg	500–1000 mg
Max. daily dosage	6000 mg	6000 mg
Dose interval (h)	4–6	6
Side effects	Liver cell necrosis due to overdose (antidote: *N*-acetylcysteine)	IV: massive drop in blood pressure, shock (no bolus injections!) Allergic reactions Perspiration Very rare: agranulocytosis
Contraindications	Caution: liver and renal function disorders	Caution: known allergy to metamizole

Non-proprietary name	***Ibuprofen***	***Diclofenac***
Preparations	Tablets/capsules 200/400/600 mg Delayed-action tablet 800 mg Suppository 500 mg Granules 400/600 mg Ampules (IM) mg	Tablets/capsules 25/50 mg Delayed-action tablet 100 mg Extended-release (resin) capsules 75 mg Dispersible tablet 50 mg Suppository 12.5/25/50 mg
Single dose	200–800 mg	50–100 mg (children 12.5 mg)
Max. daily dosage	2400 mg	300 mg
Dose interval (h)	8	8
Side effects	Gastrointestinal side effects (ulcers) Blood coagulation disorders Liver and kidney disorders Kidney toxicity Allergic reactions	See ibuprofen (stronger GI side effects than ibuprofen) at times severe anaphylactic shock and toxic epidermal necrolysis (Lyell syndrome) with parenteral administration
Contraindications	Steroid medication Known ulcers Bronchial asthma Pregnancy: strict contraindication in the last trimester	See ibuprofen Allergies

Non-proprietary name	***Celecoxib***	***Parecoxib***
Preparations	Hard capsules 100/200 mg	Ampules (IV, IM) 40 mg/2 mL
Single dose	100–200 mg	40 mg
Max. daily dosage	200 mg	80 mg
Dose interval (h)	12–24	12–24
Side effects	Dyspeptic symptoms Hypertension Heart failure Edema	See celecoxib
Contraindications	Kidney and liver function disorders Bronchial asthma Cardiovascular risk factors Pregnancy: final trimester Breast-feeding	See celecoxib

▷

Table 4.3 Overview of the Most Clinically Important Nonopioids (Cont.)

Non-proprietary name	*Flupirtin*	*Nefopam*
Forms	Capsules 100 mg Suppositories 75 mg/150 mg Ampules 100 mg	Film-coated tablets 30 mg Ampules 20 mg
Single dose	100–200 mg	30–90 mg
Max. daily dosage	600 mg	80 mg IV 270 mg oral
Dose interval (h)	8	8
Contraindications	Allergic reactions	Allergic reactions Relative: older patients, seizure disorders
Side effects	Fatigue Dry mouth Poor concentration	Tachycardia Hypertension Agitation Dry mouth Poor concentration Increased perspiration Urinary retention in older patients

Table 4.4 Interaction of NSAIDs with Other Medication: Effects Increased (Bade 1999)

The effect of these drugs is strengthened by the concurrent administration of NSAIDs:	
Oral antidiabetics	Especially with the use of salicylate. Monitor blood sugar.
Anticoagulants	Increased risk when taking salicylate, but also with NSAIDs (e. g., indometacin, diclofenac, and piroxicam). Monitor prothrombin time.
Corticosteroids	Ulcerative effect increased. Restrict use; short-term only, if any. Gastric protection (proton pump inhibitor).
Digoxin	With indometacin, diclofenac, ibuprofen. Clinical relevance unclear.
Lithium	Plasma levels and toxicity increase. Adjust dosage and monitor plasma levels.
Methotrexate	Toxicity increases. Adjust dosage.
Phenytoin	Interaction, especially with salicylates, ibuprofen, and piroxicam. Clinical relevance unclear
Potassium-sparing diuretics	Danger of hyperkalemia; monitor potassium levels.

The administration of cyclooxygenase inhibitors affects metabolic pathways so that less prostaglandin, prostacyclin, and thromboxane is produced. This in turn causes an increase in the accumulation of bronchoconstrictive leukotrienes, which explains the provocation of allergic attacks in predisposed individuals. The lack of thromboxane causes the aggregation of thrombocytes to be delayed for ~3–4 days until new blood platelets have been formed. The inhibition of the regulatory function of the prostaglandin PGE-2 in the gastric mucosa can cause ulceration, sometimes resulting in severe gastrointestinal bleeding that may even be fatal. Prostaglandins further affect the renin–aldosterone system and control the renal blood flow and the glomerular filtration rate (**Fig. 4.19**).

NOTE

The worst case scenario is that the administration of acidic antipyretic analgesics to patients with previous kidneys damage can therefore result in water and electrolyte retention and kidney failure.

The use of NSAIDs in combination with other medication can also lead to problems, as a multitude of drug interactions can occur. This should be kept in mind when treating patients who take several drugs (**Tables 4.4, 4.5**).

There are two isozymes of cyclooxygenase, COX-1 and COX-2. COX-1 is thought to be responsible for gastric protection, renal homeostasis, and the aggregation of platelets, while COX-2 is linked to inflammation, fever, and pain (**Fig. 4.20**). COX-2 has also been shown to play a significant

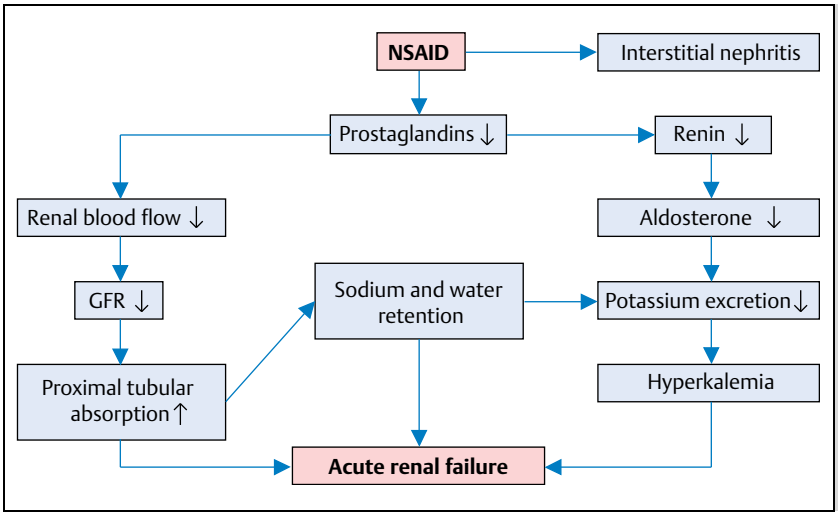


Fig. 4.19 The influence of cyclooxygenase inhibitors on the kidney and electrolyte homeostasis.

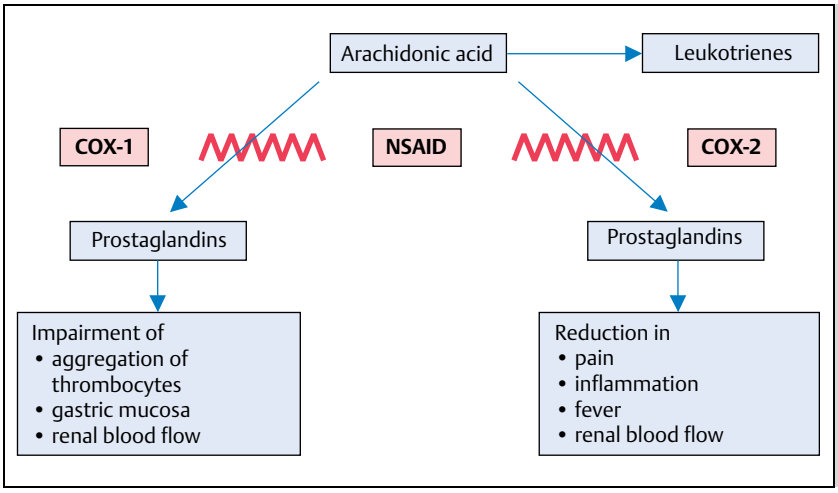


Fig. 4.20 The effect of cyclooxygenase inhibitors on prostaglandin synthesis (Yane and Boring 1995, Bakhle and Botting 1996).

role in the functioning of tissues and organs including brain, kidneys, ovaries, uterus, and endothelium.

The initial euphoria associated with the development of selective **COX-2 inhibitors** soon vanished, in the light of cumulative clinical experience and more recent research results. According to the U.S. Food and Drug Authority in July 2002, **long-term results with celecoxib** (Celebrex) demonstrated **no improvement in the gastrointestinal complication profile** compared with substances such as ibuprofen or diclofenac. Further studies have shown that the long-term use of COX-2 inhibitors, in particular high doses of rofecoxib (Vioxx 50), results in a **significant increase in the incidence of myocardial infarction and thromboembolic episodes.** Ott et al. (2003) showed in their study that the use of valdecoxib (Bextra) and parecoxib leads to a **short-term significant increase in infarction and stroke risk in high-risk patients following coronary surgery.** However, further studies at the end of 2004 proved that the **long-term use of common NSAIDs** such as naproxen can likewise play a role in the **increased rates of myocardial infarcts**. This is associated with substance-specific characteristics: the use of **sulfonamides,** in particular valdecoxib, is more frequently accompanied by **cutaneous allergic reactions** (Lyell syndrome, Stevens–Johnson syndrome) compared with methylsulfonates such as rofecoxib and etoricoxib. Etoricoxib affects kidney function and blood pressure more in the long term. Ulcers that are already present heal more poorly when patients are taking coxibs than when they are being treated with conventional NSAIDs. These findings have lead to some coxibs, e. g. rofecoxib,

Table 4.5 Interaction of NSAIDs with Other Medication: Effects Reduced (Bade 1999)

The effect of these drugs is weakened by the concurrent administration of NSAIDs:	
Aldosterone antagonists	Especially salicylate, ibuprofen
β-Receptor blocker	Antihypertensive effect is decreased

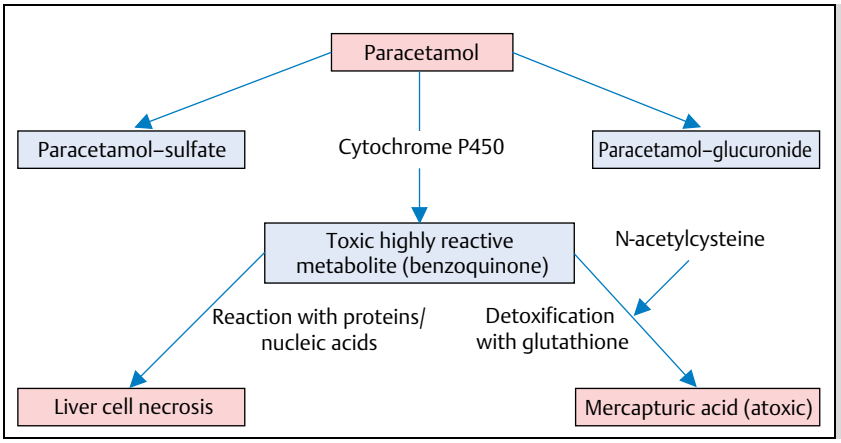


Fig. 4.21 The metabolic degradation of paracetamol.

being withdrawn from the market and not being approved by the US FDA. However, **COX-2 inhibitors** do have advantages in certain situations, e. g., in the presence of blood coagulation problems or known pseudo-allergic reactions to NSAIDs. On the other hand, NSAIDs can be advantageous when the gastrointestinal risk is low, when they are administered infrequently and, if necessary, used in combination with proton pump inhibitors. Further research into the long-term effects of traditional NSAIDs is still required to define objective criteria for patients at risk.

Nonacidic Antipyretic Nonopioids

Pyrazolone Derivatives

Metamizole, a broad-spectrum nonopioid analgesic, is the most important representative of this substance group in clinical practice. It has analgesic, antipyretic, and spasmolytic properties, and acts both peripherally and centrally.

The use of metamizole has experienced many up and downs in the past. Its side effects can be serious: In addition to the danger of a **massive drop in blood pressure,** leading to shock-like conditions when it is injected intravenously too rapidly (boluses require caution), the potentially fatal but seldom observed complication of **agranulocytosis** is feared. According to the International Agranulocytosis and Aplastic Study (IAAAS), the risk of developing agranulocytosis is estimated to be 1:1 million weeks of use (Kaufmann et al. 1986, 1991). More recent data from the Netherlands and Poland confirm these results (van der Klauw et al. 1999, Maj and Lis 2002). In 2002, a Swedish study (Hedenmalm and Spigset 2002) found a considerably higher risk (1:10 000). This paper led to renewed discussions and the withdrawal of the drug from the market in some European countries. The higher risk was contradicted by the meta-analysis of Andrade et al. (1998) in which the life-threatening complications relating to the intake of different nonopioids were compared. According to this analysis, the estimated rate of fatal episodes was 185:1 million for aspirin, 592:1 million for diclofenac, 20:1 million for paracetamol, and 25:1 million for metamizole. Ibanez et al. (2005) then confirmed the IAAAS statistics, but found a connection between the dosage level and the duration of treatment. Ibanez and colleagues came to the conclusion that in Germany the risk of agranulocytosis resulting from the administration of metamizole is very rare, and that the use of metamizole makes sense when the risk of complications with other nonopioids is taken into account. It is, however, important to note that the disposition to the development of agranulocytosis is genetically determined and that certain populations clearly react more sensitively than others. In this context, it is especially important to recognize agranulocytosis in time, with its symptoms of high fever, ulcerous lesions in the mouth and pharynx, changes in blood count, and more rarely, pneumonia and sepsis. The medication should immediately be stopped while these conditions are still reversible.

Aniline Derivatives

Paracetamol is the only aniline derivative on the market. Its mechanism of action has not yet been fully explained. It is currently postulated that there is a third form of cyclooxygenase (COX-3) that is mainly created in the CNS and is selectively inhibited by paracetamol. This could explain paracetamol's central analgesic action. In addition, its action on the central enzymatic NO synthesis and the serotonergic system has been proven.

Paracetamol can be administered to infants and during breastfeeding. The severe and, at times, fatal complication of **liver cell necrosis** has to be mentioned. This occurs as a result of overdose (more than 6 g daily over a long period of time) or glutathione deficiency (e. g., due to cachexia or pre-existing liver function disorders) and can also occur at normal dosages. *N*-Acetylcysteine is administered as the antidote (**Fig. 4.21**).

When the toxic limits of oral and especially rectal administration are taken into account, the plasma levels of paracetamol are not high enough to result in an analgesic effect, particularly in infants and young children. For this

reason the introduction of **intravenously administered paracetamol (Perfalgan)** has been welcomed in Germany. Its use enables an efficient plasma level to be reached while still keeping within the maximum daily dosage. It is important to note the **short infusion time (maximum 15 minutes)** needed to reach the desired plasma levels.

Nonopioid Analgesics without Antipyretic or Anti-inflammatory Properties

Flupirtine and Nefopam

To the best of our current knowledge, both flupirtine and nefopam act by enhancing the activity of the descending pain-inhibiting pathways. The serotonergic system also seems to be involved. Flupirtine acts additionally as a muscle relaxant and is said to interact with NMDA receptors. What this means in the prevention of chronification is under discussion, but conclusive evidence is not yet available. Documented case studies and research into chronic pain have not yet provided sufficient information, especially as flupirtine is not approved for long-term therapy because of its severe **cholestatic complications,** which should not be ignored.

Opioids

Opiate receptors are known to be present in the CNS, particularly in the limbic system, the medulla oblongata, and the dorsal horn of the spinal cord. They can also be found in the periphery, e. g., on autonomic nerves that supply smooth muscles or in inflamed tissue. Opiate receptors are subdivided into μ-, δ- and χ-receptors. They can be found both presynaptically and postsynaptically. The phosphorylation of presynaptic receptors makes the opening of voltage-dependent presynaptic calcium channels less likely to occur. The resulting depolarization causes a decrease in calcium influx and therefore reduced transmitter release. Many of the central and peripheral effects of opioids can be explained by this process.

Additionally, stimulation of μ-receptors on the postsynaptic membrane of nerve cells can cause improved potassium conductivity. This leads to hyperpolarization and therefore to decreased excitability. The inhibition of the nociceptive afferent conductivity is the foundation of analgesia.

Opioids demonstrate a diverse range of actions and side effects because of their differing affinities for the various opiate receptors. **Naloxone** is a pure antagonist and is used to antagonize opioids (e. g., to reverse respiratory depression). However, it is important to remember that naloxone has a shorter half-life than most opioids and it may be necessary to administer it several times (**continual monitoring** is required). Generally speaking, opioids are subdivided into mild (WHO level II) and strong (WHO level III) types, the latter being governed by narcotic laws (**Table 4.6**).

The opioids are further subdivided into pure agonists that only act on the μ-receptor, agonist–antagonist combinations (pentazocine, tilidine), or partial agonists (buprenorphine). The administration of pure agonists with agonist–antagonists can rapidly become unpredictable and should be avoided. It is hard to maintain a clear overview of the individual substances binding to the receptors, the timing of processes occurring, and the side effects. It is also important to remember that all weak agonists and agonist–antagonists possess a so-called **ceiling effect,** i. e., above a certain level further dose increases do not result in additional effects. An overview of the most common opioids can be found in **Table 4.7**.

Table 4.6 Potency and Receptor Behavior of the Most Important Opioids

	Agonist	*Agonist/antagonist*
Low potency	Tramadol (pethidine)	Tilidin + naloxone (pentazozin)
High potency	Morphine Methadone Piritramide Oxycodon Hydromorphone Fentanyl	Buprenorphine

Tramadol is a weakly acting pure agonist and can be used to supplement highly potent agonists such as morphine when their effect alone is not sufficient. High doses and/or too rapid administration often cause pronounced nausea and vomiting. This unwanted effect can be significantly reduced by the administration of delayed-action drugs. When the theoretical aspects of the effect of tramadol on the uptake of norepinephrine and serotonin are considered it can be seen that tramadol has a beneficial influence on neuropathic pain.

Pethidine (meperidine; e. g., Dolantin, Demerol) is metabolized into norpethidine, with a half-life up to five times longer, and can result in marked episodes of cramping when it accumulates. Renal failure as well as the induction of hepatic enzymes (e. g., by the use of phenobarbital) increase toxicity.

Although the use of **pentazocine** (e. g., Fortral) continues to spread, its objective significance in pain therapy is minimal. It acts on the autonomic nervous system, increasing pulse rates and blood pressure in the entire circulatory system, making this drug inappropriate for cardiovascular risk patients.

Valoron N contains the prodrugs tilidine and naloxone, which are enterally resorbed. Tilidine is activated to the clinically effective nortilidine via the first-pass effect in the liver and naloxone is metabolically deactivated. This does not happen when the drug is administered parentally, thus bypassing the first-pass effect, or in the presence of liver failure when metabolization is insufficient.

Buprenorphine, because of its position between level II and level III of the WHO analgesic ladder, is suitable for the

Table 4.7 Overview of the Most Clinically Important Opioids

Non-proprietary name	*Tramadol*	*Tilidin/Naxolone*
Effect strength compared with morphine	1/5–1/10	1/5–1/10
Preparations	Drops 20 drops (1 mL) = 50 mg Suppository 100 mg Ampule 50/100 mg (0.5 mL) Delayed-action tablet 100/150/200 mg	Drops 20 drops (1 mL) = 50 mg Capsule 50 mg Delayed-action tablet 50/100/150/200 mg
Single Dose	50–100 mg	50–100 mg
Single dose delayed-action preparation	50–200 mg	50–200 mg
Max. daily dosage	600 mg	600 mg
Dose interval (h)	4–6	4–6
Dose interval for delayed-action preparation (h)	8–12	8–12
Remarks	Nausea/vomiting more frequent (delayed-action drugs are better tolerated) Noradrenaline and serotonin uptake effect, eventually beneficial with neuropathic pain	Ineffective in severe liver function disorders

Non-proprietary name	*Buprenorphine*
Effect strength compared with morphine	20
Preparations	Tablet (sublingual) 0.2/0.4 mg
Single dose delayed-action preparation	0.2–0.6 mg
Max. daily dosage	2.4 mg
Dose interval (h)	8–12
Remarks	Ceiling effect Well tolerated Less constipation than with morphine Weaker development of tolerance?

Non-proprietary name	*Morphine*	
Effect strength compared with morphine	1	
Preparations	Morphine solution 0.1–4 % Suppository 10/20/30 mg Tablets 10/20 mg Ampules 10/20/100/200 mg	**Delayed-action preparations:** Tablets 10/30/60/100/200 mg Capsules 30/60/100/200 mg Suspension 20/30/60/100/200 mg
Single dose	10 mg	30–60 mg
Max. daily dosage	No limits	No limits
Dose interval (h)	4–6	8–12
Remarks	In case of severe liver or kidney failure the morphine dosage must be reduced (accumulation of M6G) Interaction with erythromycin, propofol, and cimetidine possible, leading to respiratory depression: titrate naloxone (Caution: only effective for 15 min; continual monitoring essential)	

▷

Table 4.7 Overview of the Most Clinically Important Opioids (Cont.)

Non-proprietary name	*Hydromorphone*	*Oxycodone*
Effect strength compared with morphine	7	2
Preparations	Tablet 1.25/2.5 mg Delayed-action tablet 4/8/16/24 mg	Delayed-action tablet 5/10/20/40 mg
Single dose	1.25–2.5 mg	
Delayed-action preparation	4–8 (16) mg	10–20 mg
Max. daily dosage	No limits	No limits
Dose interval (h)	As required (rescue medication)	
Delayed-action drugs	8–12	8–12
Remarks	No active metabolite → advantageous when kidney function is limited Fewer CNS side effects and less constipation than morphine Potential for dependency?	No active metabolite → advantageous when kidney function is limited Minor plasma protein binding → less interaction with high protein binding medication

treatment of severe chronic pain when symptoms are not expected to progress further. Its side effects are not as burdensome as those of morphine (constipation, nausea, vomiting) and the development of tolerance also appears to be less frequent.

Morphine is still the classic strong opioid and is currently available for almost every conceivable form of administration. Problems can occur with renal function disorders as a result of the accumulation of the active metabolite morphine-6-glucuronid (M6G), and this can lead to secondary intoxication. In these situations morphine should be administered with the utmost caution, with reduced dosages and close monitoring.

Changing over to **methadone** is often wise when patients have developed a tolerance to morphine. Because of the extremely variable half-life the time required to find the correct dosage is, however, often lengthy and difficult to manage, especially with patients suffering from renal function disorders.

Piritramide is mentioned only in passing here, as this drug, which can only be administered intravenously, belongs to the domain of acute postoperative pain therapy.

Oxycodone is a semisynthetic morphine derivative with minimal first-pass effect and correspondingly higher bioavailability (60–90%). As it produces no clinically effective active metabolites, oxycodone has an advantage over morphine in the presence of renal function disorders or extremely limited liver function. In addition, patients report less central side effects (sedation, fatigue, hallucinations) and constipation. A further advantage is that the maximum effect is reached after 1 hour (vs. 3 hours for delayed-release morphine). However, the number of case studies demonstrating the development of psychological dependency is increasing, and this is a point of concern.

Hydromorphone is a semisynthetic dihydromorphine derivative without active metabolites, which also has advantages when renal function is limited. Because of its extremely low plasma protein binding (~8%) it demonstrates less interaction with other medications that are characterized by high levels of protein binding, such as phenprocoumon (Marcoumar), NSAIDs, aspirin, paracetamol, and antidiabetics. The use of hydromorphine may be especially advisable in the treatment of elderly and/or multimorbid patients who are dependent on several drugs.

Transdermal Therapeutic System

The transdermal therapeutic system (TTS) or "patch" offers an alternative noninvasive administration route for strong opioids. In terms of the WHO analgesic ladder, **buprenorphine** is a level II–III drug and **fentanyl** a level III drug. The TTS enables a controlled, consistent release of active substances with continual absorption via the skin. It is well accepted by patients and physicians alike, as medication no longer needs to be taken several times a day. It is especially advantageous for forgetful or unreliable patients, and is also convenient for patients who have difficulty swallowing or suffer from other gastrointestinal symptoms. It is, however, a slow-acting system in terms of the uptake of active ingredients. During the first 12 hours a corresponding oral medication consisting, when possible, of the same active ingredient should be additionally prescribed. When pain additionally peaks, e. g., when weight-bearing, the use of an appropriate rescue medication is also necessary. As a general rule, the patch is changed every 3 days, although experience shows that some patients have to change the patch after 2–2.5 days. The Transtec PRO patch only has to be changed twice weekly, i. e., every 3.5 days. When using Transtec, it is

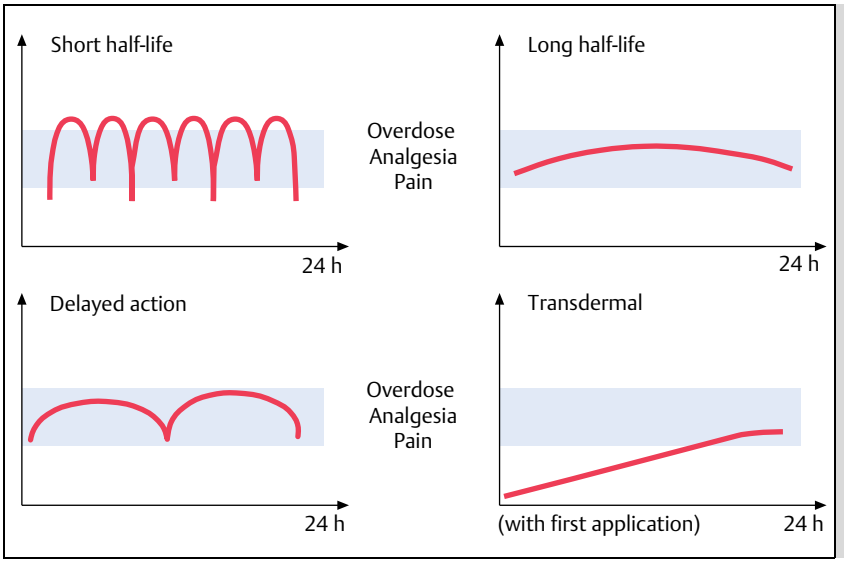


Fig. 4.22 Opioid plasma levels depending on dose intervals and half-lives.

important to bear in mind the ceiling effect of buprenorphine and the possibility of allergic–toxic skin reactions to the patch that may lead to the cessation of therapy (removal of the patch).

Opioid Management

The long-term opioid treatment of chronic musculoskeletal pain is not without controversy, for two reasons: First, this type of pain reacts badly or not at all to the administration of opioids; and second, these drugs have a considerable range of side effects. Whether the use of opioids is really justified in particular cases should therefore be carefully assessed. In addition, the suspension of opioid treatment should be attempted at specific regular intervals. If long-term opioid treatment is decided upon, a slow-release drug should be used for the basic requirements. The dose interval should be guided by the half-life of the medication (**Fig. 4.22**).

A quick-acting medication with, when possible, the same active ingredients can additionally be prescribed for breakthrough pain. When the effectiveness of the opioid progressively decreases, the dosage should first of all be raised. If the medication continues to be ineffective, or when the side effects become intolerable, a change to another opioid or an alternative administration route should be considered. The equianalgesic dose is used for guidance (see "Effective strength compared to morphine," **Table 4.7**), and it is recommended that the initial dosage be reduced by at least a third.

Opioids in the Elderly Population and in cases of Multiple Morbidity

Geriatric patients cannot always be identified simply by their age in calendar years. Biological age, multiple morbidity, and polypharmacy, as well as the threat of intellectual degradation and/or psychosocial deprivation, are much more important. It is important to bear in mind that pharmacokinetic characteristics are altered in older patients. In clinical practice, **the initial dose of opioids should be reduced by 30–50 %,** longer dose intervals should be planned, and the treatment results closely monitored, if necessary with the help of relatives. **Tables 4.8 and 4.9** provide information on the types of opioids that should be used in the presence of liver or renal failure.

Use of Adjuvant Medication in Opioid Treatment

The use of prophylactic laxatives such as polyethylene glycol (e. g., macrogol, MiraLax, Movicol), bisacodyl (e. g., Dulcolax, Laxoberal), or lactulose (e. g., Bifiteral, Enulose) is indicated as adjuvant medication. In some cases a combination of several drugs, to be taken over the entire duration of opioid treatment, may also be indicated. Antiemetics are often only required, if at all, during the initial period when determining the correct dosage. Central symptoms associated with opioid use, such as sedation, confusion, or hallucinations, should likewise be treated specifically rather than prophylactically (**Table 4.10**).

Adjuvant Analgesics

Adjuvant analgesics are especially indicated in pain syndromes that are known from experience to be only partly responsive to nonopioids and opioids. This includes, e. g., neuropathic pain, where the dose required for traditional

analgesics can be significantly reduced by the specific use of adjuvant medication (McQuay and Moore 1997, Nix 1998, Baron 2000).

Antidepressants

The use of tricyclic antidepressants is well established in the treatment of chronic pain, particularly for burning or stabbing pain. Examples of further special indications include chronic tension headaches, fibromyalgia, and postherpetic or diabetic neuropathies. Tricyclic antidepressants are assumed to act by modulating descending serotonergic and norepinephrinergic pain control systems. The effects of classic tricyclic antidepressants are the best documented, and the main effects can be divided into three types: CNS depression, antidepressive effects, and increased drive. The tricyclic antidepressants can be further subdivided into three subgroups, according to these main effects:

1. **Amitriptyline type:** These drugs act to depress psychomotor activity, repress drive, and relieve anxiety.
2. **Imipramine type:** Drive is affected neither positively nor negatively.
3. **Desipramine type:** These substances arouse and improve drive. Anxiety is, if anything, increased. There is a risk of suicide in cases of severe depression as the increase in motivation happens more quickly than the improvement in mood.

We do not yet have sufficient experience or studies of the analgesic components of the more recently developed substances such as the selective serotonin reuptake inhibitors (SSRIs). An attempt to use SSRIs can nevertheless be justified when more traditional medications are not tolerated.

Table 4.8 Recommendations for the Use of Opioids in Liver Failure. t½ = half-life, Cl = clearance, BA = bioavailability (Tegeder et al. 1999)

	Problem	*Recommendation*
Tramadol	t½ ↑ (2×)	Use with caution, reduce dose
Tilidin + naloxone	Nortilidine ↓ Naloxone BA ↑	Do not use
Buprenorphine	Insufficient data	
Morphine	Oral BA ↑ Cl, t½ ↑	Use with caution, reduce dose, (particularly oral)

Table 4.9 Recommendations for the Usage of Opioids in Kidney Failure (t½ = half-life, Cl = clearance, BA = bioavailability) (Tegeder et al. 1999)

	Problem	*Recommendation*
Tramadol	t½ ↑	Use with caution Possibly reduce dose
Tilidin + naloxone		Normal dose
Buprenorphine		Normal dose
Morphine	Accumulation of M6G (active) M3G (inactive)	Use with caution Possibly reduce dose
Oxycodone	t½ ↑ Cl ↓	Reduce dose

Table 4.10 Specific Opioid Side Effects: Frequency and Treatment Options (Freye 2004)

Side effects	*Incidence %*	*Dosage dependent*	*Tolerance development*	*Treatment*
Constipation	90–100	Yes	No	Prophylactic laxatives
Nausea & vomiting	20	No	After 5–7 days	(Possibly prophylactic) antiemetics
Sedation	2	Yes	After 3–4 days	Mostly insignificant
Confusion	2	Yes	No	Reduction of dosage Change of opioid
Hallucinations	1	No	No	Haloperidol

Table 4.11 Overview of the Most Clinically Important Adjuvant Analgesics

Non-proprietary name	***Carbamazepine***	***Gabapentin***
Preparations	Delayed-action tablet 200/400/600/800 mg	Capsules 100/300/400 mg Film-coated tablet 600/800 mg
Single dose	Start with 200 mg, increase slowly	Start with 100 mg, increase slowly
Max. daily dosage	1600 mg (monitor levels)	2400 mg
Dose interval (h)	8	8
Side effects	Initially fatigue, headache, confusion, ataxia, dizziness Changes in blood count → regular monitoring (blood count, liver, and kidney levels)	Fatigue, dizziness, light-headedness, GI symptoms, ataxia with fast loading
Contraindications	Liver functional disorders 1st and 2nd degree AV blocks Bone marrow damage	Acute pancreatitis, primary generalized seizures (no effect) Caution: Renal function disorders → adjust dose
Non-proprietary name	***Pregabalin***	***Amitriptyline***
Forms	Hard capsules 25/50/75/150/200/300 mg	Tablet 10/25 mg Delayed-action tablet 25/75 mg
Single dose	Initially 75 mg, from the 4th day 150 mg	10–75 mg
Max. daily dosage	600 mg	According to the effect, generally speaking 25–75 mg
Dose interval (h)	12	Once in the evening
Side effects	Fatigue, ataxia, coordination disorders, tremor, paresthesias dizziness, light-headedness	Fatigue Dry mouth Accommodation disorders Orthostatic dysregulation
Contraindications	Caution: Kidney functional disorder; adjust dosage	Untreated glaucoma Urinary retention Disorders of the cardiac conduction system Epilepsy

NOTE

The use of amitriptyline in pain therapy is tried and tested in clinical practice. Administered once daily at night time, it has a sleep-inducing effect that does not result in dependency, as is the case with common soporifics.

Anticonvulsants

A variety of anticonvulsants act as analgesics in the case of lancinating neuropathic pain. The drugs that have succeeded carbamazepine include **gabapentin** and the more recently developed **pregabalin**. They display significantly fewer unwanted effects, are easy to dose, and have a higher receptor affinity (**Table 4.11**).

Muscle Relaxants

Increased muscle tension is quite often associated with chronic pain syndromes. The long-term use of benzodiazepines results in dependency without benefits. When the symptoms are suitable, physiotherapy should always take priority. Treatment with pharmaceutical muscle relaxants should, if used at all, be kept as brief as possible.

Bisphosphonates

Bisphosphonates act selectively on bone metabolic resorption disorders and inhibit osteoclast activity. Their use is therapeutically indicated, e. g., in the treatment of osteoporosis and certain bone metastases.

Calcitonin

The pain-relieving effect of calcitonin (e. g., Karil, Miacalcin) is partly to do with its effect on bone metabolism, similar to vitamin D. In addition, it acts as a central anal-

Table 4.12 Overview of the Most Clinically Important Local Anesthetics

Non-proprietary name	*Lidocaine*	*Prilocaine*	*Mepivacaine*	*Bupivacaine*	*Levobupivacaine*	*Ropivacaine*
Concentration	0.5/1.0/2.0	0.5/1.0/2.0	0.5/1.0/2.0	0.25/0.5/0.75	0.25/0.5/0.75	0.2/0.75/1,.
Max. dose (mg)	300	400	300	150	150	675
Lipid solubility	+	(+)	(+)	++	++	++
Dose interval (h)	1–3	1–3	1.5–3	1.5–8	1.5–8	3–8
Significance for pain therapy	+	+	+	+++	+++	+++

gesic in the initial stages of phantom pain and the "complex regional pain syndrome" (CRPS, also known as Sudeck's atrophy).

Corticosteroids

Corticosteroids have anti-edemic and anti-inflammatory effects. They generally act via intracellular receptors. After the corticoids have bound to the receptor the complex diffuses into the cell nucleus, binds to DNA, and modulates gene expression. Given the nature of this mechanism, it is easy to understand that it takes several hours to days before the effect of the drug can be observed.

The side effects of glucocorticoids are based on the drug's main actions. Cushing syndrome occurs when the individually varying Cushing threshold has been exceeded (daily dosage of ≥ 7.5 mg prednisolone). The following side effects also occur: Ulcers in the gastrointestinal tract (corticosteroids should therefore not be used in combination with NSAIDs, if possible); increased risk of infection; delayed wound healing; atrophies of muscles, skin, and fatty tissue; danger of developing osteoporosis; diabetogenic effect; danger of developing glaucoma; increased risk of thrombosis; damage to the bone marrow; and loss of calcium. The risk rises with increased dosage and duration of therapy.

NOTE

In the special case of orthopedic pain therapy on the spine, the administration of minimal amounts of steroids in combination with local anesthetics is limited to single-shot therapy in selected cases only.

Local Anesthesia

Local anesthesia forms the foundation for diagnostic and therapeutic regional anesthesia, neural therapy, and analgesia. Local anesthetics act on the nerve endings, peripheral nerves, and spinal nerves by reversibly blocking the further transmission of cell stimulation. They do this mainly by inhibiting the influx of sodium ions, but also by partially blocking potassium and calcium channels.

Local anesthetics are an effective and low-risk addition when used correctly within pain therapy. They can be combined with opioids, nonopioids, and adjuvant analgesics at any time. The blocking of motor function is correlated with the concentration and the lipophilic nature of each drug. Side effects occur with overdoses or accidental intravascular/subarachnoid injection. CNS effects include episodes of cramping, unconsciousness, and respiratory arrest. Within the cardiovascular system, arrhythmias or ventricular blocks, even asystolia, can occur. Furthermore, there is a risk of allergic reactions and the production of methemoglobin (especially with prilocaine; see Chapter 10).

The most important characteristics of the local anesthetics currently in common use are summarized in **Table 4.12**.

II

Atlas Section

5 The Spine: Anatomy, Nociception, and the Distribution of Pain Signals

Terminology

The most common forms of acute and chronic pain originating from the spine arise from degenerative changes in the intervertebral disk and the corresponding secondary after-effects found in the vertebral motor segment. The medical conditions resulting from intervertebral disk degeneration are referred to as **spinal syndromes** and are subdivided according to their location into **cervical, thoracic,** and **lumbar syndromes.** The terms **local cervical, thoracic,** or **lumbar syndromes** are used when the symptoms are limited to the affected spinal region. When nerve root compression or pseudoradicular symptoms cause pain to radiate into the limbs, the syndrome is labeled **cervicobrachialgia** or **sciatica** of the lumbar spine. In the thoracic spine, the previous familiar name of intercostal neuralgia has been replaced by the term **thoracic root syndrome.**

All of the biomechanical and pathological anatomical changes in the intervertebral segment related to intervertebral disk degeneration are known as **degenerative disk diseases.** These include a loss of central pressure and the development of fissures and signs of wear, resulting in a loosening of the intervertebral disk. **Spondylosis** and **osteochondrosis** are bony reactions in individual vertebral parts. They are not a diagnosis; rather, they are simply a radiographic symptom of a loosened intervertebral disk.

The displacement of intradiscal mass with the formation of sequestra can be observed in intervertebral disk degeneration. When deciding which therapeutic approach to use, it is essential to identify whether the posteriorly displaced intervertebral disk tissue is only a **protrusion,** where the anulus fibrosus is still intact, or whether the **sequestrum has moved outward** as a result of the perforation of the outer border of the disk, taking the form of a **prolapse** or **herniation.**

Nociception and the Distribution of Pain Signals in the Spine

The individual nociceptive complexes are closely packed together within the spine: mechanical insults coming from the intervertebral disk, nociceptors in the posterior longitudinal ligament and zygapophyseal joint capsule, nociceptively changed afferent fibers in the spinal nerve, and the distribution of noxious signals in the spinal cord. The medical conditions observed, the so-called spinal syndromes, are correspondingly complex. Types of pain and spread appear that can be linked to the irritation of the different parts of the nociceptive system. Neuralgias occur where pain is projected into and felt in the area of distribution of a segmental nerve; the cause of this disorder is not to be found in the area of pain. Brachialgia and sciatica are typical examples of this. Neuralgia is differentiated from organ pain by its position: in organ pain, the location of the pain and the disorder itself are identical, as in the local cervical and lumbar syndromes.

In addition there are forms of pain, especially in the cervical spine, where sympathetic nervous system impairment also plays a role. In this case, the pain is constant and diffusively spread, and is associated with vasomotor trophic symptoms. Pain that is linked to a 24 hour rhythm or a hormonal cycle, or that occurs frequently in autonomically labile individuals, is likely dependent on the sympathetic nervous system. Despite a multitude of symptoms that cannot be precisely defined, most types of pain coming from the spine can be linked to a certain neural structure within the vertebral motor segment.

Pain-Sensitive Structures in the Vertebral Motor Segment

Human intervertebral disks do not posses nerve fibers. Sensitive nerve endings have so far only been found on the outermost edge of the anulus fibrosus at the posterior longitudinal ligament (Kuhlendahl 1950, Kuhlendahl and Richter 1952, Mulligan 1957, Mendel et al. 1992). These histological examinations have been experimentally confirmed by Smith and Wright (1958). During surgery they fastened thin nylon threads to different vertebral motor segment structures and to the nerve root. After the operation was completed, typical symptoms could be induced

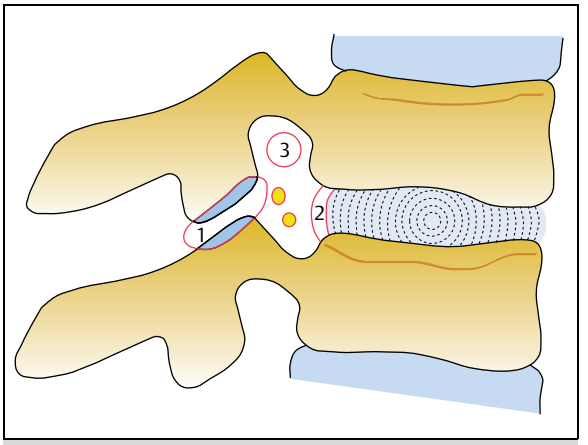


Fig. 5.1 Pain-sensitive structures found in a vertebral motor segment. Zygapophyseal joint capsule (1), posterior section of anulus fibrosus and posterior longitudinal ligament (2), irritated spinal nerve root (3).

by pulling on the posterior anulus fibrosus and on the nerve root.

Kuslich and Ulstrom (1990) stimulated the different tissue structures in the vertebral motor segment during a lumbar intervertebral disk operation performed under local anesthetic, and registered the pain sensitivity. They found that pain could most easily be provoked on the skin and on the compromised nerve root, followed by the outer anulus fibrosus and the posterior longitudinal ligament. The ligamental attachments and the zygapophyseal joint capsules were less often sensitive to pain. The ligamenta flava, lumbar fascia, lamina, facet joint cartilage, and noncompromised nerve roots were completely insensitive to pain (**Fig. 5.1**).

Anterior intervertebral disk puncture, e.g., during a cervical discography, is pain free, just as lateral puncturing during a lumbar discography is pain free. Pain and feelings of pressure are not felt during the administration of a contrast agent when the intervertebral disk is intact. When the intervertebral disk is protruding and already in contact with the nerve root, an increase in volume can result in a spreading pain. This is used diagnostically in the distension test. During a medial transdural lumbar disk puncture, patients report only a short, lumbago-like pain sensation in the back when the posterior longitudinal ligament or the posterior anulus fibrosus is punctured. In lumbar disk operations under local anesthetic it is also possible to elicit typical back pain by using a probe to place pressure on the posterior edge of the intervertebral disk.

Spinal Nerve

The spinal nerve consists of motor, sensory, and sympathetic components. After passing through the intervertebral foramen it divides into ventral and dorsal rami and a meningeal branch.

The **ventral ramus** is the most extensive. It supplies the anterior regions of the body and the limbs and is responsible for the dermatome-oriented nerve root syndrome.

The **dorsal ramus** supplies the skin and muscles of the back. In addition, it has extra branches running to the outer surface of the zygapophyseal joints and their capsules. The paired posterior branches can be seen alongside the vertebral column from the occiput to the coccyx. They pass through the fascia so as to reach the areas of skin that they supply. They are found relatively near the midline in the thorax (T1–T11). In the lumbar region (L1–L5) they are found lateral to the muscular bulge of the spinal extensors, and in the sacrum (S1–S4) paramedian over the posterior foramen.

The **meningeal branch** re-enters the spinal canal and then branches, supplying the inside section of the zygapophyseal joint capsule, vertebral periosteum, posterior longitudinal ligament, and the spinal cord meninges with efferent, afferent, and autonomic fibers (**Fig. 5.2**). The area surrounding the intervertebral foramen is a **likely trouble spot for the development of pain in the vertebral motion segment.**

This is especially the case in the lower sections of the cervical spine and lumbar spine. Here the vertebral foramina are enclosed by the zygapophyseal joints as well as the intervertebral segment; nociceptors and afferent fibers are found close to each other. The afferent fibers can develop into nociceptors when chronically irritated. A mechanical irritation of the sensitive fibers of the meningeal branch can occur in the zygapophyseal joint capsule, in the posterior longitudinal ligament, and in the spinal nerve root itself. Pain is induced by the displacement of intervertebral disk tissue with pressure on the posterior longitudinal ligament or on the spinal nerve root. These types of pain are described as primarily discogenic, or as symptoms originating from the intervertebral disk. In contrast, secondary discogenic pain originates from the zygapophyseal joints or from the back muscles.

NOTE

In the mechanical irritation of nociceptors and afferent fibers, the amount of deformation is not important. What matters is how quickly the deformation happens.

Even torsion scolioses, where the spine deviates considerably from the body's axis, are symptom free when they have developed over many years. Nerve roots, ligaments, and zygapophyseal joint capsules are obviously able to adapt. On the other hand, a small posteriorly protruding intervertebral disk can cause the most severe symptoms when it appears suddenly and makes even minimal con-

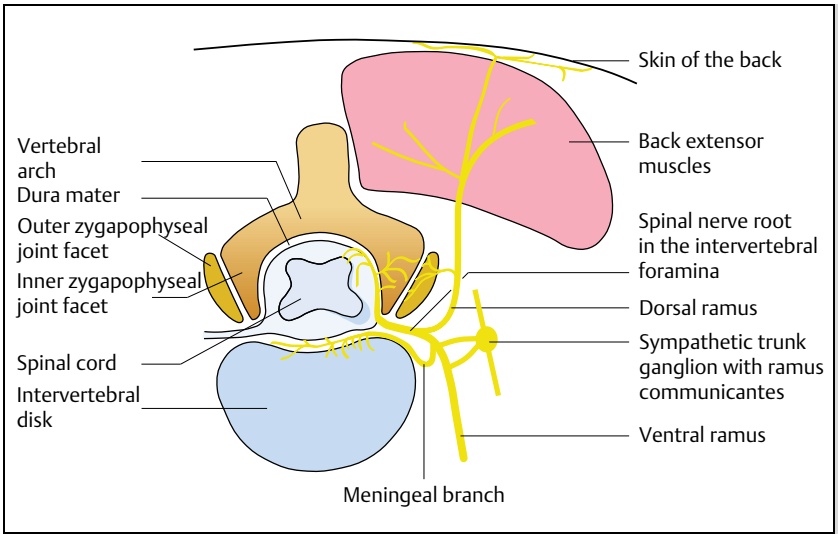


Fig. 5.2 The spinal nerve and its branches: Ventral ramus, dorsal ramus, meningeal branch. The meningeal branch and the ventral ramus are connected to the sympathetic trunk ganglion via the rami communicantes.

tact with the pressure-sensitive nociceptors in the posterior longitudinal ligament or in the nerve root.

Posterior Longitudinal Ligament

Pain originating in the posterior longitudinal ligament or the posterior anulus fibrosus is dull and difficult to localize. Pain can appear suddenly and severely, as in the case of lumbago or a wry neck, or gradually, as when a severe kyphosis or abnormal increase in intervertebral volume places great tensile stress on the posterior intervertebral disk. The meningeal branch of the spinal nerve and its corresponding nociceptors are considered possible carriers for the vertebral column's own pain sensitivity. It cannot be said with absolute certainty whether or not the mechanical compression of the dura acts as a source of pain. Large medial prolapses and spinal tumors do not cause pain.

Nerve Root

The spinal nerve root is by far the most vulnerable and irritable part of the neural conductive pathway. The compression and stretching of nerve tissue are the basic mechanical factors that play a role in the initiation of disease. Mechanical irritation takes place only along the preganglionic path of the nerve root and is mainly due to pressure and elongation of the meningeal or mesenchymal tissue, causing irritation and corresponding sensations of pain. This results in a wide variety of presenting symptoms. The **radicular syndrome** has the following characteristics:

- Pain radiates along the dermatomal distribution bands.
- Sensory disorders present solely as sensitivity to pain.
- Muscle atrophy is isolated and does not correspond to a peripheral nerve.
- The loss of reflexes does not pertain to the peripheral nerve that appears to be affected.
- There is no sign of disturbances to the autonomic nervous system.

The spinal nerve root fibers innervate the skin and muscles of the trunk and limbs, as well as the vertebral column itself. For this reason a variety of combined symptoms are possible. In particular, the symptoms may be segmentally ordered when the ventral ramus is mainly affected. When a spinal nerve root is compromised, the affected segment can be identified by specific sensory and motor disorders in the trunk and limbs. The presenting symptoms may be more prominent in one spinal nerve branch, depending on the position and severity of the spinal nerve root irritation (**Fig. 5.3**). All types of pain combinations can be seen within the segmentally defined boundaries. The composition, quality, and quantity of pain changes with the amount of pressure on the nerve root. All levels of pain are possible, from pure pain radiation (the so-called bands of pain), without objective signs, up to complete anesthesia. Investigations by Smith and Wright (1958) demonstrated that the extent of radiating pain distribution is proportional to the amount of pressure on the nerve root. In other words, minimal contact causes sciatic pain radiating only down to the thigh, for example, while more severe compression causes radiation down to the foot.

The **discogenic nerve root compression** can be influenced by altering the volume and consistency of the intervertebral disk and changing the position of the vertebral motor segment. Many diagnostic and therapeutic measures used for intervertebral disk-related symptoms are based on this fact. There is normally sufficient room between the dural sac, the exiting nerve root pairs, and the posterior border of the intervertebral disk to allow spinal movements, changes in the contours of the intervertebral disk, and changes in nerve root position to a certain extent

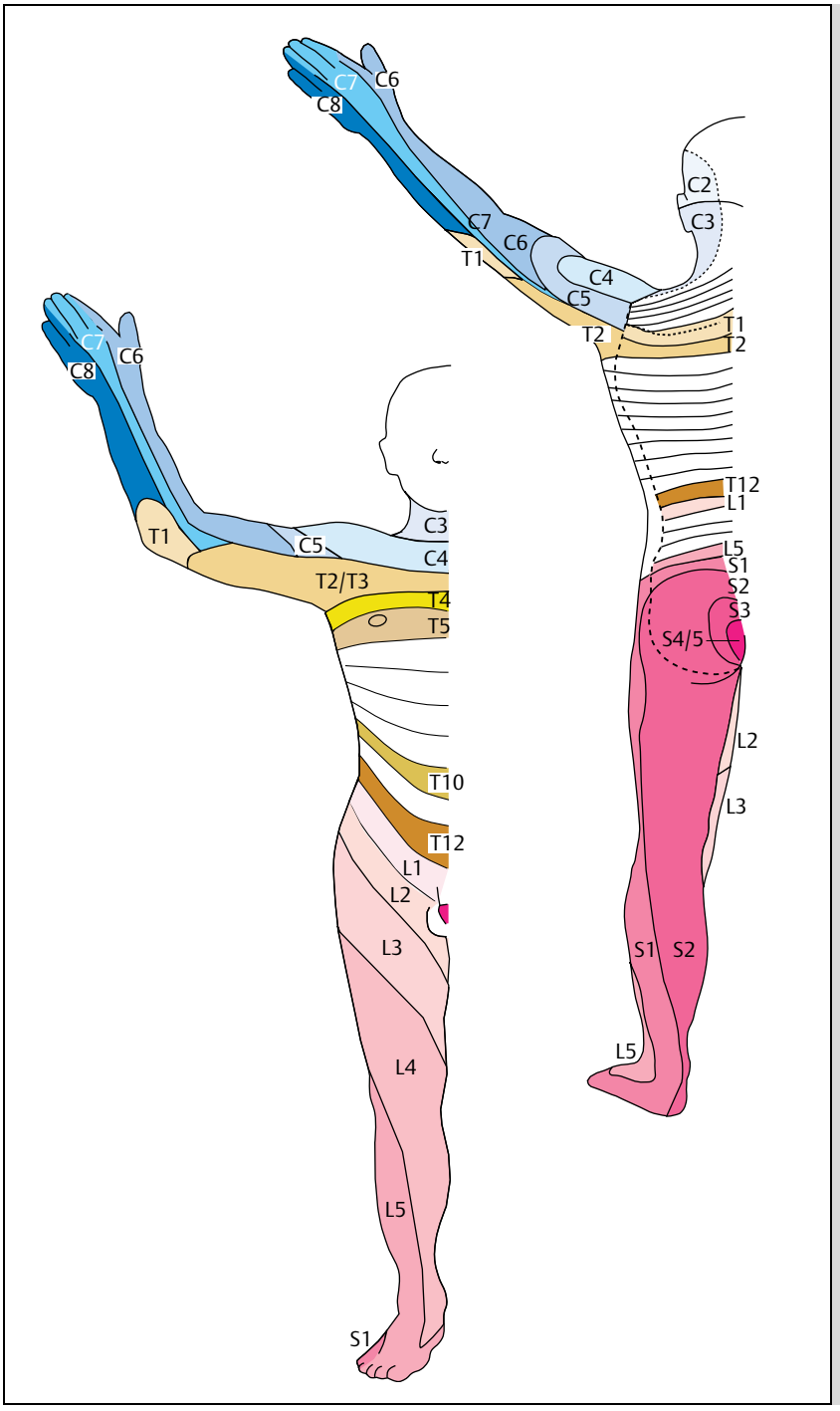


Fig. 5.3 Dermatomal spread arising from the ventral ramus of the spinal nerve.

without pressing in on the spinal nerve. The spare space between the dura, nerve root, and intervertebral disk is filled with loose fatty tissue and a venous plexus. Its width varies according to the individual spinal segment. There is also normally sufficient room between the nerve and the bony borders in the intervertebral foramen.

When the space surrounding the spinal nerve is filled up with intervertebral disk protrusions, osteophytes, thickened vessels, or a narrowing of the spinal canal (spinal canal stenosis), the nerve root is compressed by even the slightest of provocations. When the nerve root comes into contact with the surface of the intervertebral disk, the physiological, pressure-dependent fluctuations in consistency and volume are passed on to the spinal nerve. The nerve is no longer able to adjust its position, and reacts sensitively to certain spinal movements. This explains the changeable characteristics of intervertebral disk-related symptoms.

An **osteogenic nerve root compression** is also possible, in addition to the discogenic root compression. The prerequisites for this are found especially in the cervical spine, where osteophytic reactions occur, radiating out from the uncinate process. In the lumbar spine, arthrotic osteophytes can also be found on the zygapophyseal joints as well as in the form of spondylitic processes on the posterior edge of the vertebral body. All of these can compress the nerve root. The pain of osteogenic nerve root compression is characterized by its persistence and resistance to conservative treatment measures. Also, it can be well localized as the same part of the nerve is always being irritated.

A **biochemically induced nerve root irritation** is provoked by the displacement of intervertebral disk tissue. This tissue is not normally found in the spinal canal, and its presence there induces a foreign body reaction. Saal and Saal (1989), Olmarker and Rydevik (1993), and Willburger and Wittenberg (1994) experimentally proved the direct toxic action of the intervertebral disk tissue on the nerve tissue. In the case of a disk prolapse, inflammatory reactions along with mechanical impingement of the nerve root are decisive in the development of pain.

The mechanical or chemical irritation of a spinal nerve root causes microscopic changes in the nerve root. The nerve root either swells up with edema, or atrophies after long periods of compression. Shades of red or blue livid discoloration can sometimes be observed during surgery. **A nerve that has been damaged by compression develops nociceptive characteristics in the course of chronification.** Spontaneous action potentials develop, and the amount of excitability changes in axons that have been demyelinized as a result of compression (Wehling 1993).

A nerve root irritated by inflammation is much more sensitive to touch stimuli than it is in its normal state. This can readily be confirmed by touching the irritated nerve root during disk prolapse surgery. Many treatment measures, in particular local infiltration, aim to reduce the oversensitivity of the nerve root and to desensitize it.

Nociceptive Pain Arising from the Zygapophyseal Joints

Pain arising from the spinal nerve and its branches, with the exception of the meningeal branch, displays neuralgic characteristics: i. e., it is a radiating pain. In contrast, the pain arising from zygapophyseal joints results from the activation of nociceptors in the zygapophyseal joint capsule. The pain is felt at the location of the pain source, i. e., in the lower lumbar spine.

NOTE

Zygapophyseal joints are a highly significant cause of pain in the vertebral motor segment because they have such large numbers of receptors.

Joint capsules, and the joint facet synovium and periosteum, have mainly free nerve endings along with some (Vater–Pacini type) encapsulated nerve endings.

Like all joints of the body, the zygapophyseal joints are designed for specific movements by means of the positioning of their cartilage surfaces and the stretchability of the capsule. The initial and final positions of the zygapophyseal joint are determined by the corresponding intervertebral disk. During contrast imaging and our attempts at compression we have observed that the amount of joint play in the zygapophyseal joint is rather large. A sudden excursion past the physiological end point is called a ligament strain or tear. Occasionally a **blockage** occurs and the joint remains in a pathological end-position.

These conditions—especially when they occur suddenly—are always associated with pain originating from the mechanonociceptors in the joint capsule, which are sensitive to pressure and tensile stress. A decrease in intervertebral segment height, such as in intervertebral disk degeneration, is associated with a change in the initial position of the associated zygapophyseal joints. Under these circumstances, spinal movement within the normal range can cause capsule-stretching pain as the joint's endpoint is exceeded. Repeated malpositioning or permanent loading results in arthrotic changes. Unlike primary discogenic symptoms, zygapophyseal joint arthrosis is associated with **deep, dull pain** that does not immediately react to changes in posture or extension (traction) (**Figs. 5.4 and 5.5**). Ghormley (1993) described pain arising from the zygapophyseal joint capsule as the facet joint syndrome.

NOTE

The facet joint syndrome is a nociceptive type of pain that has to be correspondingly identified and treated.

Muscles

Shoulder, neck, trunk, and hamstring muscles can be affected by pathological processes in the intervertebral segment in two ways. First, there is the possibility of incorrect or even painful permanent innervation when the dorsal ramus of the spinal nerve is irritated. Second, the trunk and proximal limb muscles compensate for instabilities and can become overstrained. In this way, endogenous and exogenous pain stimuli develop that act on the nociceptors in the vertebral motor segment. Painful muscular tension is found especially when joint capsules are irritated. Muscle tension is partially controlled by the receptors in the joint capsules. A spontaneous pain that increases with pressure and movement develops in the affected muscle. In this way irritation in the lower lumbar zygapophyseal joints, for example, results in reflex pain in the lumbar back extensors, the buttocks, and the posterior leg muscles. The muscle pain, running from the origin to the insertion, results in arm or leg pain. The sensitivity to pressure, with radiation into the painful areas of the limbs,

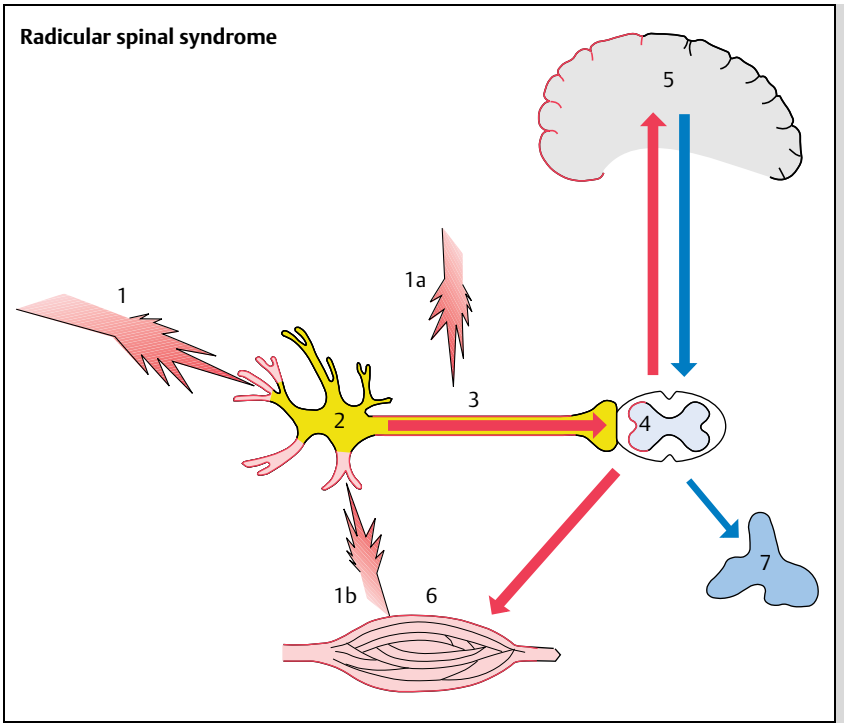


Fig. 5.4 Radicular spinal syndrome with chronic neuralgic pain. The noxious stimulus (1a) acts directly on the nerve (3) which has changed into a nociceptor. The motor reaction (6) with increased muscle tension produces endogenous noxious stimuli (1b), which in turn stimulate the nociceptors, and a component of nociceptor pain is produced.

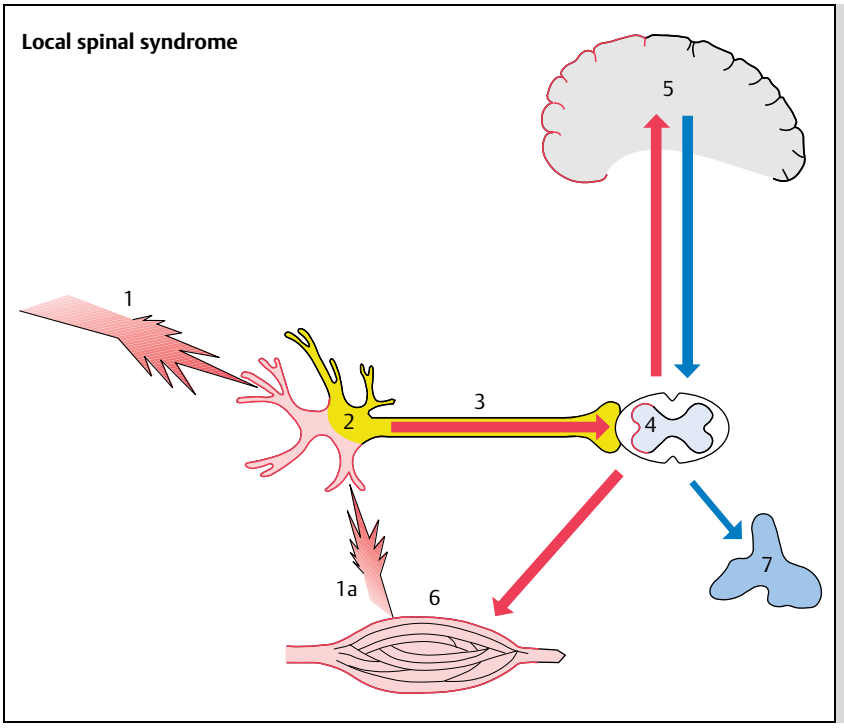


Fig. 5.5 Local spinal syndrome with nociceptor-defined acute and chronic pain. The pain stimuli (1) act on the nociceptor (2). Transmission of signals occurs in 3 and 4, including a motor reaction (6) that in turn acts as an endogenous noxious stimulus (1a), affecting nociception (2) within the vicious circle.

confirms the pseudoradicular character of the symptoms (**Fig. 5.6**). **The specific administration of local anesthesia to the affected zygapophyseal joint is helpful in reaching a confident diagnosis.** Such pseudoradicular nociceptive-related pain sensations are either the forerunner of an intervertebral disk protrusion or the residual symptoms following surgical or conservative treatment of a disk prolapse.

Mixed Pain Syndromes in the Spine

In vertebral motor segments the source of pain and the afferent fibers are close to one other. Different combinations of symptoms can occur here, because the motor, sensory, and autonomic components of nerve fibers are linked.

A segment is identified as the area supplied by the spinal nerve. This includes the dermatome as zone of influence, the sensitive nerve fibers in the skin, and the myotome as the area of influence for motor spinal nerve fibers within the skeletal muscle. Disorders in the somatic and autonomic parts of the ventral ramus of the spinal nerve have repercussions on the vertebra itself, the functional area of the meningeal branch, and the dorsal ramus. It is only in rare cases that isolated pain appears. In most cases symptoms are mixed, and continue to alter during the course of the disease. The development of symptoms with the involvement of all nerve branches and nociceptors occurs especially when nociceptors and afferent fibers are chronically irritated, e. g., within the scope of a postdiscectomy syndrome (**Table 5.1**).

NOTE

When a vertebra compresses the nerve root, pure radicular symptoms are first seen followed by a mixture of pain types with radicular, pseudoradicular, and tendomyogenic components.

Chronification of Pain Arising from the Vertebrae

Acute pain that originates in the vertebral column is generally nociceptive pain, as described above. This pain comes from the posterior longitudinal ligament and posterior anulus fibrosus, e. g., it is initiated by a displacement of intradiscal mass (lumbago). A zygapophyseal joint or a sacroiliac joint can be temporarily pathologically malpositioned when the intervertebral disk is no longer stable. Pain occurs due to stretching of the capsule, as previously discussed. The corresponding nociceptors are sensitized when repeatedly irritated.

The afferent section of the dorsal ramus and the meningeal branch lie very close to each other. This allows neuralgias to appear during the course of the disease. The muscles and ligaments associated with that segment are secondarily involved with the additional nociceptive

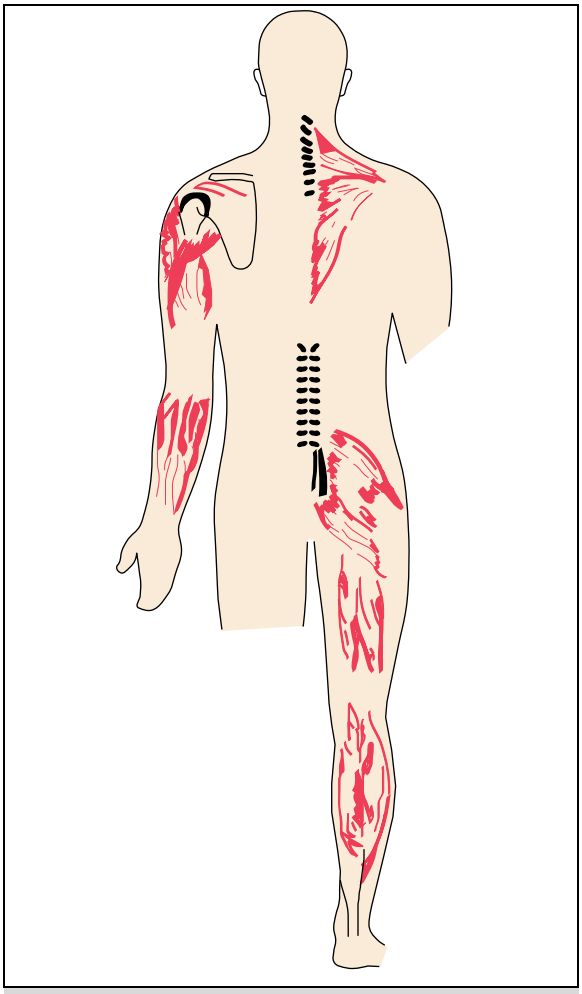
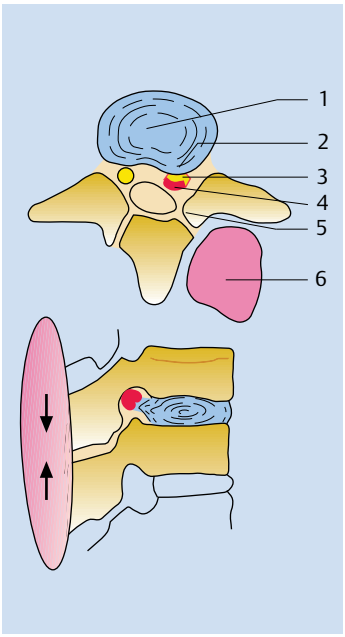


Fig. 5.6 Nonradicular radiation into the extremities in the form of pseudoradicular pain, radiating out from the vertebral motor segment. The pseudoradicular pain in the shoulder/cervical muscle region radiates from the cervical zygapophyseal joints or, as the case may be, from the shoulder joint. Sciatica-like symptoms in the hamstring muscles originate in the lumbar zygapophyseal joints and the sacroiliac joints.

Table 5.1 Nociceptors, Afferents, and the Character of Chronic Vertebral Pain

Nociceptor	*Afferents*	*Pain character*
Posterior longitudinal ligament, posterior anulus fibrosus	Meningeal branch	Nociceptive pain
Spinal nerve	Ventral ramus, dorsal ramus, meningeal branch	Neuralgia + nociceptive pain
Zygapophyseal joint	Dorsal ramus, meningeal branch	Nociceptive pain
Muscles, ligaments	Dorsal ramus	Nociceptive pain



	Noninvasive	Minimally invasive/surgical
1. Expansion pressure in the disk	Fowler's position, traction	Interosseous therapy
2. Intervertebral disk prolapse mass	——	Discectomy, open and percutaneous, chemonucleolysis
3. Inflammatory contact, disk prolapse, root	Analgesics, anti-inflammatories	Epidural injection, neurolysis
4. Inflammatory root swelling	Analgesics, anti-inflammatories	Epidural–perineural injection
5. Malpositioning of the zygapophyseal joint, stretching of joint capsule	Fowler's position, flexion orthosis, physiotherapy	Facet infiltration, fusion
6. Reflex spasm in back extensors	Muscle relaxants, heat treatment, massage, electrotherapy	Paravertebral injection

Fig. 5.7 The development of chronic vertebral pain and the methods used for causal and symptomatic orthopedic pain therapy.

pain. The vicious circle of chronic pain arising from the vertebrae is maintained by

- chronically irritated nociceptors in the posterior longitudinal ligament, the posterior anulus fibrosus, the zygapophyseal joint capsule, muscles, and ligaments
- nerve fibers in the ventral ramus, dorsal ramus, and meningeal branch of the spinal nerve that have changed into nociceptors.

The coexistence of limited movement in the vertebral column, tense muscles, radicular and local symptoms, associated autonomic symptoms, and psychological reactions justifies a multimodal approach to orthopedic pain therapy. The choice and sequence of treatment modalities used is based upon the most predominant component of pain.

Pain arising from the vertebrae is accompanied by a multitude of psychological changes as the duration of illness increases. Psychological components generally play an important role earlier and more intensively in the permanency of spinal symptoms than they do with pain in the joints or limbs. It is therefore crucial to pay attention to the corresponding diagnostic clues when these types of symptoms are present. When pain arises from the vertebrae, the earlier the psychological components of chronification are prevented, the better the prognosis (**Fig. 5.7**).

6 Special Orthopedic Injection Therapy: Contraindications and Patient Information

Contraindications

As with all local injections, inflammatory bacterial changes around the injection site (e. g., infected sebaceous glands) and open wounds are contraindications for spinal injection therapy. Systemic infections that are either acute or have only recently subsided (e. g., urinary tract infections, tonsillitis, common colds) should also be absent. When such infections are clinically suspected, further diagnostics should exclude these infections before starting on the planned injection therapy.

Because of the possible danger of infections occurring or flaring up again, it is important to ensure completed secondary healing following surgey on the intervertebral disk, epidural abscesses, and spondylitis.

Known hypersensitivity to local anesthetics is another contraindication for minimally invasive spinal therapy. Any suspicion that substances will provoke an allergic reaction should be checked in advance.

The general contraindications to the use of steroids should also be kept in mind. The side effects of glucocorticoids are related the main actions of the drug (see Chapter 4, "Multimodal Medication Concomitant Therapy") and the administration of a single dose does not usually cause dangerous adverse effects. When the medical history shows that patients who require long-term treatment have conditions such as osteoporosis, diabetes, glaucoma, or ulcers, IRAP treatment, for example, can be used as an alternative to steroids (see Chapter 4, "Interleukin-1 Receptor Antagonist Protein").

Orthopedic injection therapy is also contraindicated for patients with neurological seizure disorders, spinal cord diseases, or severe cardiovascular disorders because it may require intrathecal administration, with sudden drops in blood pressure. Any injections near the spine are contraindicated for patients with internal diseases such as severe conduction defects, decompensated heart failure, or blood coagulation disorders.

Patients who are taking anticoagulants such as Warfarin (Coumarin) should be put on low-dose heparin before minimally invasive spinal therapy is undertaken. The intake of thrombocyte aggregation inhibitors such as aspirin or ticlopidine (Tyklid), is also a contraindication. Patients should be taken off this medication at least a week before having any injections near the spine, in consultation with the general practitioner.

Table 6.1 Contraindications for spinal injection therapy

Local and/or systemic infections
Open wounds
Known hypersensitivity to local anesthetics
Steroid contraindications
Neurological seizure disorders
Spinal cord diseases
Severe cardiovascular disorders
Severe conduction defects
Decompensated heart failure
Blood coagulation disorders
Concurrent blood-thinning medication (aspirin, coumarin, etc.)
Absence of the prerequisites for injection therapy (emergency equipment, monitoring facilities, dedicated injection room, etc.)

The environment plays an important a role in keeping the patient calm (Grifka et al. 1999). The treatment room should be a sterile environment, suitably equipped for all of the injection therapy procedures (Geiss 2002, Mutter et al. 2002).

After therapy patients should be specially monitored, so that any disorders of vital functional can be recognized in time and prevented. Equipment for venous access, oxygen supply, intravenous drips, and resuscitation must be available in case injection-related complications arise (**Table 6.1**).

Patient Information

The physician's duty to provide information to the patient is founded on the professional code of conduct. From a legal point of view, every type of medical intervention used for diagnoses or treatment potentially represents illegal bodily harm. The patient's consent is required to justify the intervention. Patients have the right to be informed in an understandable and complete manner, during a personal discussion with their treating physician, about the clinical picture of their symptoms and the interventions proposed for diagnosis and treatment. Information can be divided into three categories, relating to diagnosis, disease progression, and risks. Information about the diagnosis is preparation for explaining the treatment itself. The explanation of disease progression should inform the patient about the likely further development of symptoms if the intended intervention is carried out, and if it is not. Alternative treatment possibilities should be described at this stage. An explanation of the risks involved, i. e., the probability that the intervention will be unsuccessful and the possible consequences of the planned treatment, is central to in the physician's duty in providing information.

Informative discussions, recorded in writing, are established clinical practice. Drawing a simple sketch to describe the planned intervention can dramatically improve patients' understanding of the intended therapy. The sketch can be added to the consent form.

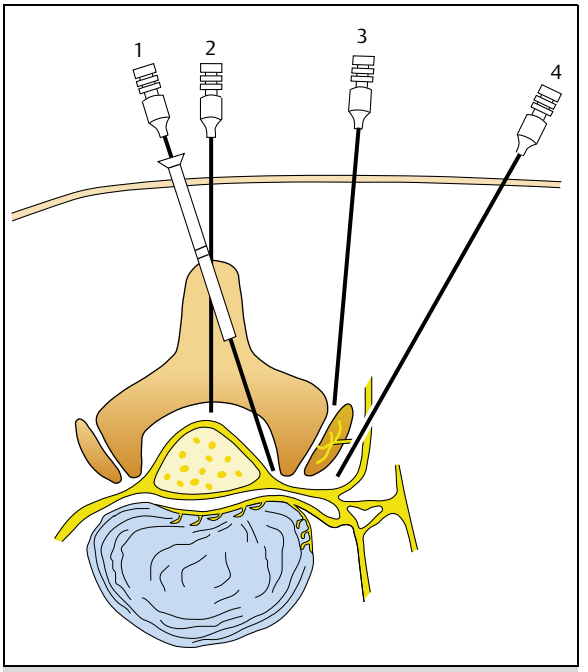


Fig. 6.1 Schematic illustration of the cross-section through a vertebral body. The most common injection techniques are clearly shown: epidural perineural (1), posterior epidural (2), periarticular (3), paravertebral perineural (4).

As well as discussing the general risks, the specific risks associated with each individual type of injection must also be mentioned. Many patients are wary or fearful of injection therapy, for a whole range of reasons. During the discussion the doctor should attempt to abolish any fears that the patient may have, and convince them that the intended injection therapy is the most appropriate treatment option.

Standardized informed consent forms have been in use for many years. However, these forms merely complement the personal individual discussion; they do not replace it. The standard forms should be regarded as containing the basic information that the patient needs. They should be designed to allow room for any individual additions that may arise during the personal discussion with the patient. In case of doubt, a third party should be able to see clearly that the patient's questions have been sufficiently explained and that patient was given enough time to understand the intended form of treatment.

As already mentioned, the informed consent form can be complemented by a simple sketch showing how and where the intended injection will be performed. A schematic illustration of the cross-section of a vertebral body clearly shows the most common injection techniques (**Fig. 6.1**).

Injury to blood vessels and nerves is one of the typical side effects and complications generally associated with spinal injections. Injury to blood vessels mostly results in harmless minor bleeding, but in isolated cases injury to larger vessels can result in more extensive bleeding and the development of a hematoma. On principle, patients should be questioned as to whether they take blood-thinning medication and this should be documented. Injury to nerves can lead to temporary or permanent disorders associated with pain, sensory disturbances, movement disorders, functional disorders in the affected inner organ, and paresthesia of individual muscles and muscle groups. The complications are mostly harmless and of short duration. In most cases special medical treatment is not required.

Despite meticulous sterile procedures that adhere to the relevant hygiene standards, the administration of medication directly into the source of pain can result in inflammation, mainly caused by bacteria. Surgical intervention may be necessary and some functional losses may remain. In extremely rare cases bacteremia and life-threatening sepsis may occur, especially when indwelling catheters are used.

In individual cases, the administration of analgesics can result in altered levels of consciousness, respiratory disorders, and gastrointestinal side effects in the form of

nausea and vomiting. These are mostly of short duration and generally require only short-term monitoring. Allergic reactions to the local anesthetic, injected medication, preservatives, and contrast medium are also possible. The symptoms can range from general allergic reactions, such as skin rashes, itchiness, and nausea, up to life-threatening anaphylactic shock. Patients should therefore be asked whether they have any known allergies before starting on the injection therapy. In case of doubt, preliminary testing of the intended medication is necessary. Symptoms that arise from allergic reactions can also result from the accidental intravascular injection of medication. In severe cases intensive medical treatment may be necessary (see Chapter 10).

One of the special risks associated with spinal injection therapy is possible injury to internal organs. Injury to the lung with consequential pneumothorax is primarily possible when injecting into the lower cervical, thoracic, and upper lumbar spines. Epidural injections in the cervical, thoracic, and upper lumbar spinal areas can also lead to spinal cord injuries. The consequences include permanent functional disorders of the affected nerve stem and the internal organs supplied by the nerve, ranging up to paraplegia. Injections into the lumbar spine can injure the kidneys and the efferent urinary tract. Injury to internal organs is rare, but can nevertheless be severe and require surgical intervention. The administration of local anesthetic can lead to temporary paralysis of individual muscles and muscle groups, with the danger of falls.

When using roentgen rays, e. g., when injecting with imaging or with CT-guided injections, the exposure to radiation must be mentioned to the patient. Pregnancy must be excluded.

The patient should also be informed of the possibility that the presenting symptoms will remain even though the injection therapy is correctly carried out.

NOTE

Despite its possible side effects and complications, spinal injection therapy is one the most fast-acting and effective methods available in orthopedic pain therapy.

7 Cervical Injection Therapy

Specialized Cervical Neuroanatomy

Chronic cervical pain arises primarily in the lower cervical vertebral motor segments from C5 to T1. One reason for this is that the strongest pathological anatomical changes are found here, because of the exceptional loading placed on the point of curvature at the cervico-thoracic junction. Another reason is that the spinal nerves, sympathetic plexus, and vertebral artery are found immediately adjacent to disorders of form and function within the vertebral motor segment.

Chronic headaches and dizziness also often arise from the head–neck junction. These are caused by joint capsule irritation, functional disorders, and deformations in the area of the atlanto-occipital and atlanto-axial joints.

There are seven cervical vertebrae and eight cervical spinal cord segments. As a result of developmental displacement, vertebral motor segments and spinal cord segments are not always found at the same level. The amount of displacement increases from cranial to caudal and is already clearly apparent in the lower cervical segments. In the lower cervical region, the spinal cord segments are found one vertebra higher up in relation to the spinous processes. The spinal nerve roots from C4 and downward run in an inferior and lateral direction until they pass through the intervertebral foramina. Segmental syndromes are named according to the affected spinal nerve root, the number of which also corresponds to the inferior vertebral body in the damaged vertebral motor segment. For example, in a C6 syndrome the C5/6 intervertebral disk is affected, in a C7 syndrome the C6/7 intervertebral disk and so on. The C8 nerve root exits via the C7/T1 intervertebral foramen (**Fig. 7.1**).

The precise identification of the affected segment is important for local treatment, cervical spinal nerve analgesia, and surgery. In the last few years it has been possible to determine the individual area of supply of each spinal nerve root in a purely empirical manner, as a result of discography, distension tests, and the many surgical interventions now being used in the diagnosis and treatment of cervical syndromes. Cervical-related nerve root irritation syndromes present mainly in a monoradicular form. Overlapping and mixed symptoms are possible when anastomoses are present under the spinal nerve, found partially inside the dural sac, and during the concurrent irritation of two or more nerve roots.

The C6, C7, and C8 nerve roots are most commonly affected. The dermatomes associated with nerve root irritation syndromes, radiating as far down as the hand, overlap in the upper arm and forearm (**Fig. 7.2**). All dermatomes have a posterolateral band of pain and paresthesia in the shoulder and upper arm in common. In the forearm, the C6 syndrome radiates more laterally and the C8 syndrome more medially. The C7 syndrome is found between the two (see **Fig. 5.3**).

Cervical Sympathetic Chain

The vertebral artery and the cervical sympathetic chain are found in the immediate vicinity of the uncovertebral region in the lower cervical vertebral motor segments. This is

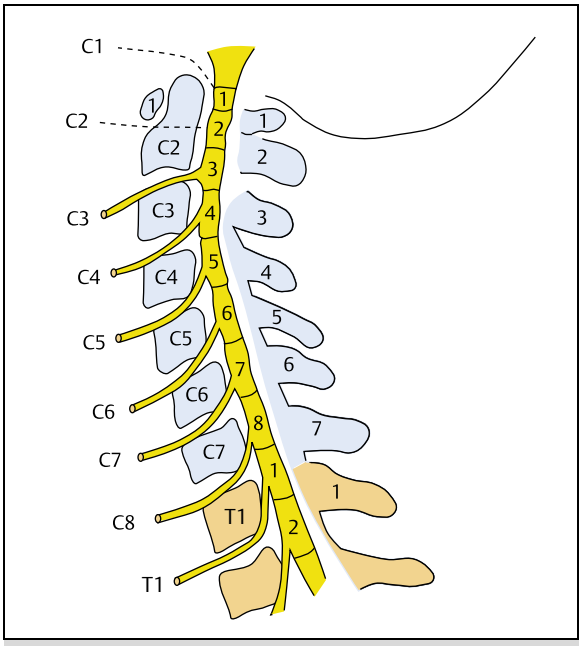


Fig. 7.1 Cervical spinal nerves and nerve roots. The spinal nerve roots from C4 downward run in an inferior and lateral direction until they pass through the intervertebral foramina. For example, the C5/6 intervertebral disk is affected in a C6 syndrome and the C6/7 intervertebral disk in a C7 syndrome.

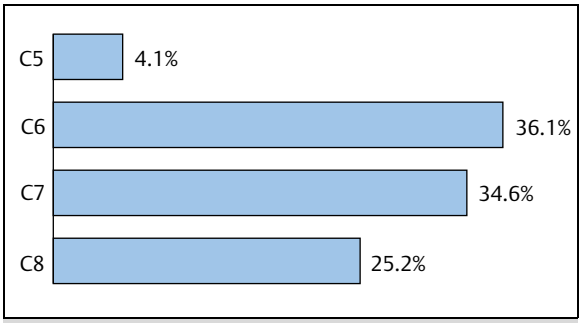


Fig. 7.2 The distribution of monoradicular CBS.

of particular clinical importance, as the close relationship explains why both can be affected during the process of aging and are considered possibly responsible for a complex neurovascular set of symptoms. The cervical sympathetic trunk is connected to the spinal nerves via the gray ramus communicans. It has three cervical ganglia responsible for the autonomic innervation of the head/neck region and the upper limbs. The upper ganglion encompasses the C1–C4 segments, the middle ganglion the C5–C6 segments, and the lower ganglion merges with the uppermost thoracic ganglion to form the stellate ganglion, encompassing the C7–T2 segments. The stellate ganglion is especially important as a major distributor. All efferent and almost all afferent sympathetic fibers coming from the head, neck, arm, and upper thorax run through this ganglion. Fibers travel cranially from here, including via the sympathetic arterial plexus of the vertebral artery.

Studies by Hovelacque (1925), Wrete (1934), Kummer (1984), Kehr and Jung (1985), and Bogduk and colleagues (1988) have called attention to the entanglement of the cervical sympathetic chain, the cervical spinal nerve, and the vertebral artery. Sympathetic nerve fibers travel from all three cervical ganglia of the sympathetic trunk to the C4–C8 spinal nerves and partially encompass the vertebral artery (**Fig. 7.3**).

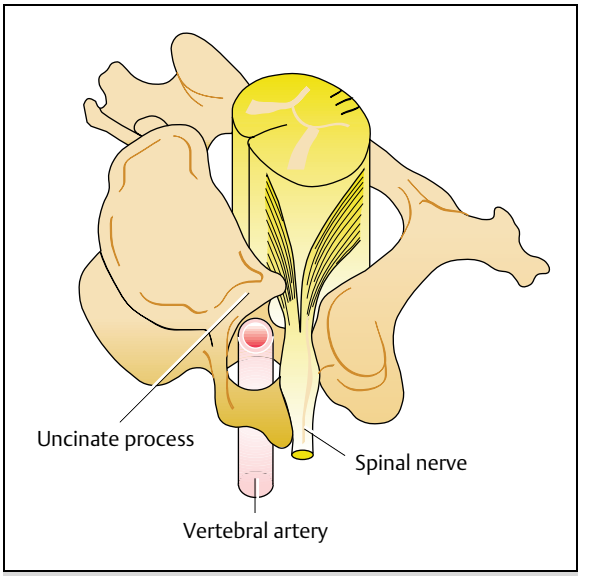


Fig. 7.3 The topographical relationship between the uncinate process, the vertebral artery, and the spinal nerve. Degenerative expansion of the uncinate process presses in on the spinal nerve and the vertebral artery, resulting in concurrent irritation of the sympathetic nervous system.

Basic Therapy for Cervical Pain

A wide range of methods is used to treat pain originating in the cervical spine. **Polypragmasy** is permitted during pain therapy because of the complex symptoms presented in the cervical syndrome. It is not the primary mechanical components alone that have to be treated: treatment is also required for secondary symptoms such as increased muscle tension, postural problems, and psychological changes. Causal and symptomatic treatment methods are administered in parallel.

When degenerative changes are already present, mechanical events are the primary cause of cervical syndromes. These events include sudden changes of position, hypermobility, and the protrusion or bending of intervertebral disks or bones. **Causal therapy** should always aim to combat one of these pathogenetic components. Secondary symptoms, such as increased muscle tension, postural problems, and psychological changes, must be treated alongside the primary mechanical components. Heat treatment, electrotherapy, massage, and analgesics should eliminate these secondary symptoms and disrupt the vicious circle of muscle tension caused by pain leading to more pain due to adaptive postures.

An important component of the treatment of cervical syndromes is the temporary use of a **cervical collar,** especially in the acute phase. Cervical collars prevent all movements that cause repeated mechanical irritation of the already irritated nerve root, the sensory receptors in the posterior longitudinal ligament, and the zygapophyseal joints from the first phase onwards. The neck posture can involuntarily cause pain during careless movements or when sleeping. In addition, the insulating collar accumulates body warmth in the shoulder/neck area and relaxes the muscles there.

The use of **physiotherapy exercises** is especially advisable during the rehabilitation phase. When there is pain in the cervical spine, isometric muscle-strengthening exercises for the shoulder and neck muscles are appropriate first-line treatment. Wide ranges of movement should be avoided, so that nerve roots and nociceptors in the zygapophyseal joint capsules are not irritated further. All exercises—as with all concurrent forms of treatment—should start with a flexed cervical spine. Approximately 10–15° of flexion unloads neural elements to the greatest extent. This is especially the case with the intervertebral foramen and the zygapophyseal joint capsule (Krämer 2009).

Manual therapy primarily involves the use of manipulation with axial traction (**Fig. 7.4a, b**). When cervical manual therapy is primarily directed toward the zygapophyseal joint the thrust also affects the intervertebral disks, because the zygapophyseal joint and the intervertebral disk form a functional unit within the vertebral motor segment. Brief, firm traction forms the basis for almost all manual therapy techniques on the cervical spine and results in a reduction in intradiscal pressure. This in turn, has a suction effect that pulls any laterally or posteriorly displaced intervertebral disk tissue back into its original position. It is important, however, to consider the contraindications.

In causal pain therapy cervical traction is carried out either manually or with the help of traction devices. Many types of apparatus are available for cervical traction, some more expensive than others. Gentle traction, easily measured, is possible with the use of a Glisson sling. Its use in clinics and practices is well established (**Fig. 7.5**). The sling must fit the head well, with no pressure being applied to the chin or the larynx. In traction of the cervical spine the correct positioning of the patient is also important, just as it is in physiotherapy treatment. Glisson traction of the cervical spine is usually carried out with the patient lying supine. Less force is required in this position than in a sitting position, as the weight of the head is eliminated. The direction of pull should not be along the body's axis, but rather more anterior, in terms of a slight cervical kyphosis, and when possible in the direction of the relieving posture.

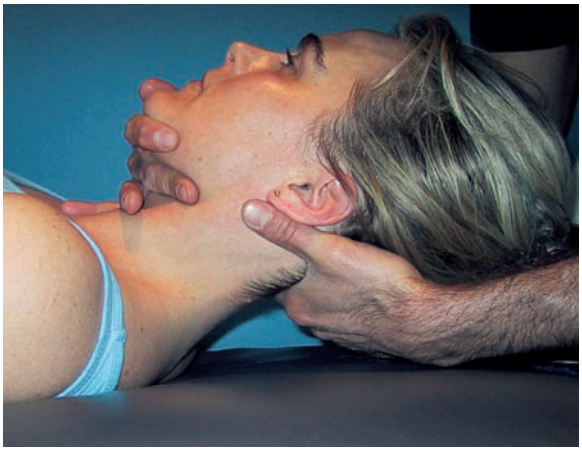


Fig. 7.4a, b Manual therapy for the treatment of pain originating in the cervical vertebral motor segments: Neural elements are relieved by traction in a neutral position (**a**) or in the direction of relief (**b**).

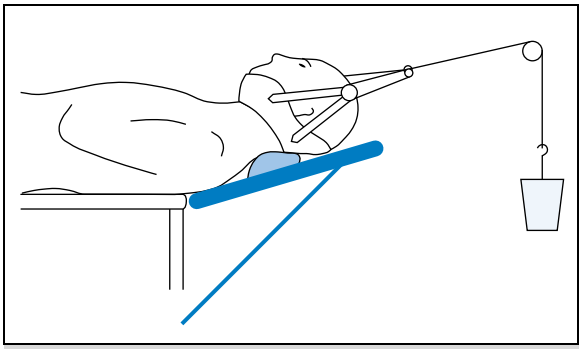


Fig. 7.5 Glisson kyphosis traction performed on the cervical spine as causal pain therapy.

Behavioral Guidelines for Patients with Cervical Pain: Back School

The rules of back school also essentially apply for patients with recurring pain in the shoulder/neck region. Rule 1 is important: To keep moving the neck and to avoid remaining in the same posture, especially when the position is unfavorable. The range of movement should not be too large, however, in order to avoid unnecessary irritation of the nerve root and zygapophyseal joint capsule. Activities where the posture and line of vision remain constant should be interrupted as often as possible. This especially applies to reading, watching television, working on a computer, and driving. Movements that aggravate pain, such as cervical extension and rotation, should be avoided at all

costs, especially when chronically recurring pain is present. (It is particularly hard to avoid rotary neck movements while parking a car parallel with the curb. If they have to do this, patients should turn their whole body as much as possible, and use the rear view mirrors.) The remaining behavioral guidelines are essentially orientated toward maintaining optimal neck posture with mild flexion. The behavioral guidelines for patients with cervical pain are summarized in **Table 7.1**.

The behavioral guidelines also apply to sporting activities. Generally speaking, back-friendly types of sport—swimming, jogging, cycling—are also recommended for patients with chronic cervical problems. Even in these types of sport a lot depends on the position of the head. For swimming, backstroke with a posture that relieves loading is recommended. When jogging, the head should be held with the chin down and neck lightly flexed—not held back with the neck extended, as some joggers do. The same applies for cycling: The handlebars should be set high enough that the neck is slightly flexed. Needless to say, swimming, jogging, and cycling can be practiced during back school under professional supervision.

In order not to further stimulate irritated cervical nociceptors and nerve roots, the only permissible movements are those that that allow a constant, nonvibratory, relieving neck posture in slight flexion. The best exercise is stationary cycling, ideally in front of a television screen placed to ensure a good head/neck position. Backstroke in a neck-relieving posture can also be permitted in the early stages of acute and chronic cervical syndromes.

Table 7.1 Back School Rules for Chronic Cervical Pain

1	Take short breaks when reading, working with your hands, watching television, or driving.
2	Do not turn your head abruptly—it is best to turn the entire body.
3	Do not let a draft blow on your bare neck—wear a scarf or collar.
4	Use a small pillow when lying down. Do not lie on your stomach.
5	Keep your chin down when jogging, handlebars up when cycling.
6	Do not work above your head. Use a ladder or a chair instead.
7	Do not sit in the front row at the theater or cinema. It is best to sit at the back.
8	Use a straw when drinking out of a can or bottle.
9	Wash your hair under the shower, not leaning over the washbasin.
10	Do daily neck muscle exercises.

Special Therapy for Cervical Pain

Local Cervical Pain Syndrome

By definition, local cervical syndromes are limited to the neck area, i. e., there is no radiation into the arms or head.

NOTE

In the local cervical syndrome, pain is felt at its origin. **The clinical picture is therefore governed by nociception.**

The nociceptors in the zygapophyseal joint capsules, the posterior longitudinal ligament, and the uncinate process are affected. The nociceptors at the cervical muscle insertions and the muscles themselves are secondarily involved. The causes are unstable segments and functional disorders in the cervical vertebral motor segments.

The pain is felt only in the shoulder/neck region and alters with changes in position, tense muscles, and limited mobility in the cervical spine. The symptoms can begin suddenly, e. g., when the head is abruptly turned, or they may increase gradually without a specific cause. During the subjective assessment, patients often mention chill and the effects of drafts among the precipitating factors. The patient can quite clearly localize where their pain is coming from during the examination. When the cranial cervical segments are affected, the pain lies within the area of supply of the dorsal ramis along the upper edge of trapezius, running from the occiput to the acromioclavicular joint.

When the lower cervical motor segments are irritated, a **neuralgic-type component** may also be present if **pain between the shoulder blades** is mentioned. This is mediated by the dorsal ramus. As well as the typical tender points, there is some noticeable tension in the entire shoulder and neck musculature and cervical mobility is limited. When an acute pain syndrome is present, an immediate motor reaction occurs and the patient adapts their posture with a slight amount of **cervical flexion and head rotation. This posture should not be changed,** as it represents a **protective reflex** which aims to prevent further irritation of the nociceptors.

Pain Therapy for Acute Local Pain Syndromes

Pain should be treated immediately to prevent it becoming chronic. Peripherally acting analgesics block nociceptors and the distribution of pain signals at the source of pain. This treatment primarily involves the use of NSAIDs administered per os or as an infusion, when necessary. At the same time, local infiltration of the zygapophyseal joints, facet infiltration, and the infiltration of muscle insertions at the occiput, the edge of the scapula, and the acromioclavicular joint is recommended. The application of cold in

the acute phase and heat for more enduring symptoms can be used in addition. Depending on the main focus of symptoms, these interventions can be supplemented by manual therapy, with traction in the direction of relief; the temporary use of a cervical collar so as to avoid neck positions that aggravate pain, especially at night; concomitant psychological therapy; and postural and behavioral training (back school) starting as soon as possible.

All methods are justified that relieve the patient's acute pain and do not disrupt the patient's normal way of life, i. e., patients should be able to sleep and to carry out activities of daily living, even if they may be slightly restricted. From this point of view, it is obvious that all methods that generally sedate the patient (central analgesics) or depend on bed rest are inappropriate. Interventions that affect the nociceptive region locally are the focus of attention.

Treating physicians should use the methods that they are most familiar with, so long as they fulfill the prerequisites mentioned (**Table 7.2**).

Table 7.2 Pain Therapy for Acute Cervical Syndrome

Analgesics (NSAIDs)
Cervical collar (at night)
Cold (heat)
Local infiltration (trigger points, facet infiltrations)
Manual therapy (traction)

Table 7.3 Pain Therapy for Chronic Cervical Syndrome

Heat
Local injections
Movement therapy
Back school
Progressive muscle relaxation
Exercise program

Pain Therapy for Chronic Local Pain Syndromes

The indication that **pain is becoming chronic** is that it endures for weeks or months. Patients are unable to rest, especially at night. This in turn soon results in the development of associated psychological symptoms. The character of the pain changes, and as the disorder progresses further it is constantly present. Initially the pain is precisely located at the occiput and between the scapulae. It later develops into a diffuse pain that extends over the entire shoulder/neck region. The constant tension in the trapezius, levator scapulae, and rhomboid muscles causes insertion tendopathies at the occiput, the upper border of the scapulae, and the acromioclavicular joint. As the shoulder is secondarily involved in this process via the proximal muscles of the upper limb, chronification of pain can also occure here, and may even become an independent pain disease. The cervical sympathetic trunk is irritated at the uncovertebral region and acts as the primary mediator for the autonomic reaction, which takes the form of local paresthesias, circulatory disorders, and feelings of warmth/cold.

The clinical symptoms and the associated psychological symptoms change when the cervical syndrome pain is present for weeks or months on end. The chronic pain becomes the focus of attention and requires preferential treatment. Therapy primarily includes the use of heat treatment, exercises, and local infiltration at the sources of pain. Analgesics should be administered sparingly when chronic pain is present.

The primary aim of treatment for a chronic cervical syndrome is to break the vicious circle of pain–cramped muscles–adaptive posture–pain. Originally, the increased muscle tension was useful; however, it becomes independent via the vicious circle as the chronically recurring cervical syndrome progresses. This results in pain caused by constant muscle tension and insertion tendopathies (**Table 7.3**). Massage, electrotherapy, infiltration of muscles using local anesthetics, and general muscle-relaxing measures are ideal for breaking the vicious circle at the muscular level. Progressive muscle relaxation according to Jacobson (1938) is the primary intervention.

Exercise Program

The exercise program for chronic cervical pain syndromes includes all movements that occur distant to the cervical spine and, if possible, do not cause axial vibration in the vertebral column. Suitable activities include stationary cycling, swimming in a neck-relieving posture, and all exercises carried out supine or in a reclined sitting posture. Jogging and road cycling are not initially recommended because of the vibrations associated with these activities. The same applies to housework and gardening, as involuntary quick cervical extension and rotation movements take place during these activities.

All types of sport that involve movement above head level must be treated with caution: e. g., tennis, volleyball, badminton. It is possible to start playing tennis by initially playing at the baseline without serving overhead, for example. Sport physiotherapists can provide specific instructions.

Psychological Components of the Chronic Cervical Syndrome

Several psychological factors act together in chronic cervical pain. The disorder is in close proximity to the CNS, which makes patients very concerned and anxious. **Catastrophic thinking** arises, for example, with thoughts of permanent disability and the threat of paraplegia. These thoughts are reinforced when physicians show patients

radiographs of bony growths or—even worse—MRI images with posterior protrusions which are pressing on the spinal cord or may do so in the future.

The **physician has an important role** in the initial stages by explaining the link between pathology and anatomy, using a model when possible, pointing out that the disorder is only temporary and the outcome positive when the spine becomes stiffer with age. The physician must make it clear that degenerative changes in the cervical spine, such as protrusions and spondylitic growths, almost never result in paraplegia. The cervico-medullary syndrome with spinal cord compression is a quite exceptional degenerative spinal disorder and **should not be mentioned** to patients with chronic cervical syndromes.

On the other hand, **trivializing** or ignoring pain and pain-aggravating factors is also inappropriate. The sensitized nociceptors in the shoulder and neck region are irritated by even the slightest of inappropriate postures in extension and rotation. When patients wake up in the night with their neck involuntarily positioned in a hyperlordotic and rotated posture, the primary problem is not a psychological one. Stress modulates the vicious circle of pain and muscle contraction, playing an important role in the chronic cervical syndrome. Patients often report that their pain intensifies when they are stressed at work or in their private life. They feel more pain in the neck muscles, and more superiorly in the form of a tension headache. The increased frequency of neck pain and headaches, combined with the patient's reduced ability to cope with stressful situations, often leads to an increasingly depressive mood. To prevent or disrupt this development, the patient should begin a muscle relaxation program before it is too late.

When the pathological muscle tension has eased, the use of exercises is appropriate when they are carried out carefully and in the pain-free range of movement. Postural and behavioral training during back school dictate that all movements and postures that can result in new nociceptive input must be completely avoided. In chronic cervical syndromes this is especially important for cervical extension and rotation movements. Chronic pain may continue to develop when the shoulder/neck region is exposed to cold or in the presence of psychological stress.

Pain Therapy for the Cervicobrachial Syndrome

Radiating pain in the arm that can be allocated to a specific dermatome indicates the presence of a **neuralgic clinical picture.** The ventral ramus of the spinal nerve is primarily affected. Cervicobrachial syndrome (CBS) is provoked either by an intervertebral disk prolapse or by hard, bony constrictions at the uncinate process. Bony constrictions occur significantly more often. The combination of segmental instability and uncovertebral osteophytes is of clinical importance.

The **clinical symptoms** generally appear gradually and are governed by a dermatome-related brachialgia that is position dependent. The afferent fibers in the spinal nerve have to first be converted to nociceptors. Night-time pain that is accompanied by a feeling of numbness and ants crawling along the dermatome is characteristic of this disorder. A chronically recurring progression is typical. The syndrome can be accentuated by external factors (e. g., acceleration injuries of the cervical spine) and a constantly maintained unfavorable posture (e. g., working at a desk, watching television).

The C6 syndrome involves pain radiating down to the thumb. In some cases the biceps tendon reflex is weakened. Radiation into the middle finger with possible weakness in the triceps and the triceps reflex, combined with thenar muscle atrophy, is characteristic of the C7 syndrome. In the C8 syndrome, pain radiates along the medial side of the hand. Sometimes a motor disorder of the finger flexors and the muscles of the hypothenar eminence may be present.

CBS is a primary chronic disorder. The conversion of a primary conducting nerve to a nerve with a nociceptor function already represents chronification.

The longer the condition remains, the less chance there is that simple interventions such as manipulation, traction, or a nerve root block can improve the pain rapidly. The constant nerve irritation leads to secondary symptoms with motor and autonomic reactions. Over time, central changes occur in the perception and processing of pain. In some cases, characteristics such as different positions affecting pain levels and the day–night pattern are no longer seen: Pain is felt permanently. Associated psychological symptoms appear. As in the local pain syndrome, chronification noticeable in the secondary muscle tension, insertion tendopathies, and the maladaptive postures found in the shoulder/neck/arm region.

The pain therapy used to treat CBS is not just directed toward the peripheral nociception. It aims, in addition, to affect the transmission and processing of pain signals. **Local injection treatment with cervical spinal nerve analgesia is central in pain therapy for CBS.**

It makes sense to directly tackle the source of pain at the intervertebral foramina or the uncovertebral region in acute as well as chronic cervical root irritation syndromes. To stop chronification, or interrupt the process at the spinal nerve root, daily spinal nerve analgesia is required for the first 10 days (including weekends). After the injection, patients should be placed in a position that relieves the affected part of loading for at least 30–60 minutes, ideally using Glisson traction. Further treatment is supplemented by physiotherapy with exercises starting in the pain-relieving position, electrotherapy, heat treatment, and progressive muscle relaxation exercises. The local injection treatment includes not only cervical spinal nerve analgesia at the affected segment, but also the infiltration of secondary sources of pain, e. g., at the border of the scapula, the occiput, and the deltoid muscle. The use of acupuncture is also established in the treatment of CBS.

Outpatient and inpatient treatment plans are described in Chapter 13.

Treatment of Cervical Headaches (Cervicocephalic Syndrome)

A cervicocephalic syndrome (CCS) is a cervical syndrome accompanied by headache, dizziness, and sometimes dysphagia, hearing, and visual disorders. Headaches can be provoked by the stimulation of nociceptors in the zygapophyseal joints, neck muscles, and muscle insertions.

Possible sources of irritation include malpositioned joints at the head/neck junction, deviations of the cervical spine from the body axis, displacement of vertebrae, and constriction of the vertebral artery by lateral bony growths on the uncinate processes of C4–C7. The convergence of nociceptive afferents in the dorsal horn of the spinal cord or in the inferior trigeminus nerve nucleus can cause radiating facial pain, especially in the forehead, eye, and temple regions. The main **clinical symptoms** are persistent, chronically recurring headaches. The headaches are neuralgic in nature and have a chronic character right from the start. Predominant symptoms include headaches and dizziness that are position dependent, being worse when the neck is extended and rotated, in addition to the symptoms associated with the local cervical syndrome. This disorder is also described as **cervical migraine** because of the presence of severe headaches. In addition to the migraine-like ipsilateral headaches which radiate into the forehead, bilateral neuralgic headaches with an ipsilateral emphasis located at the back of the neck are also observed (**Table 7.4**).

The type of therapy to be used is dictated by the etiology and pathogenesis of the cervical headache. Headaches originating directly from the head itself are primarily treated by the administration of analgesics. In contrast, cervical headaches can be relieved simply by a change of posture and slight flexion of the neck. This position is adopted as the starting position for further physiotherapy and manual therapy interventions. Heat treatment to stimulate the circulation in the shoulder/neck region, electrotherapy, and progressive muscle relaxation are also used.

The source of pain is in the uncovertebral region of the lower cervical vertebral motor segments, so the use of cervical spinal nerve analgesia, with 8–10 injections at C7 and C8, is recommended. When the headache is felt more on one side, the injections are administered on the affected side. In the case of symmetrical headache, the injections are administered alternately on both sides.

A suitable exercise program conducted within the pain-free range of cervical movement is important, combined with the appropriate postural and behavioral training taught in back school. The inpatient and outpatient treatment plans used for CBS also fundamentally apply to the treatment of headaches arising in the cervical spine (see Chapter 13).

Table 7.4 Characteristics of Cervical Headaches

Unilateral emphasis
Posture dependent
Episodic, short-term appearance
Local cervical syndrome symptoms are present at the same time

Pain Therapy for the Posttraumatic Cervical Syndrome

An important application in pain therapy is the treatment of acceleration injuries of the cervical spine, often known as whiplash injuries.

NOTE

The symptoms arising from an acceleration injury of the cervical spine are identified as a **posttraumatic cervical syndrome (PTCS).** Whiplash is only one of the possible causes of symptoms. PTCS can arise from any type of force that causes comparatively severe bending or compression of the cervical spine.

The cervical spine is a relatively weak link between the head and the thorax, being free to move in practically all directions. Traumatic compression and bending of the cervical spine occurs during sporting activities, such as handball and boxing, and in other situations, such as fairground rides or amusement parks.

Overextension of the cervical spine occurs when the trunk is fixed and the head is strongly accelerated in a posterior direction. The nociceptors in the zygapophyseal joint capsule become highly irritated, especially in the lower cervical vertebral motor segments. In some cases, nerve roots are compressed inside the intervertebral foramina. This results in shoulder and neck symptoms, sometimes with radiation into the arm. It is characteristic of PTCS that patients experience a pain-free interval between the injury and the onset of the first symptoms. A posttraumatic cervicocephalic syndrome then develops, with symptoms such as persistent headache at the back of the head and occipital neuralgia.

The use of cold packs, immobilization, and analgesics is central to **pain therapy** in the treatment of acute PTCS. A strong tendency for pain to become chronic is seen especially when claims for damages can be filed based on the injuries caused by the trauma. It is therefore important that the cervical collar be removed as soon as possible, and that exercises are prescribed. The use of manipulation with large of ranges movement is not appropriate, as this would maintain the instability in the vertebral motor segment caused by the initial distortion trauma.

Cervical Injection Therapy

Cervical Spinal Nerve Analgesia

Principle

Posterolateral injection of a local anesthetic, combined with steroids when necessary, into the foraminal articular region of the lower cervical vertebral motor segments.

At the point where the cervical spinal nerve exits the intervertebral foramina of C5/C6, C6/C7, and C7/T1 it is possible to reach not only the exiting spinal nerve, but also the meningeal branch as it returns into the spinal canal and the dorsal ramus that runs to the zygapophyseal joint capsule and the segmental back extensors. Despite the external form of administration, the nociceptors found in the posterior longitudinal ligament, the posterior anulus fibrosis, and the zygapophyseal joint capsule can be indirectly reached via the meningeal branch. Cervical spinal nerve analgesia (CSPA) thus affects discogenic (meningeal branch), arthrogenic (dorsal ramus), and radicular (ventral ramus) pain in the cervical vertebral motor segment (**Fig. 7.6**).

Table 7.5 Indications for CSPA

Cervical root syndrome
Cervicocephalic syndromes
Post-traumatic cervical syndrome
Pseudoradicular cervical syndrome

Indications

Indications include C5, C6, C7, and C8 cervical nerve root syndromes. Further indications are cervicocephalic syndrome, posttraumatic cervical syndrome, and local cervical syndromes with severe symptoms (**Table 7.5**).

Technique

Posterior entry, 3–4 cm paravertebral, enables the physician to reach the lower cervical spinal nerves and parts of the cervical sympathetic chain at an average depth of 3–6 cm craniolateral to the most lateral bony border, without running the risk of dural puncture or injury to the large neck vessels.

Cervical spinal nerve analgesia is performed with the patient in a sitting position, with maximal flexion of the cervical spine and the arms hanging down by the side. First of all, the injection site is marked. The C5, C6, C7, and, when necessary, T1 spinous processes are used for orientation, and a longitudinal line connecting these points is marked. The injection site lies 3–4 cm lateral to the marked medial line and is found halfway between two spinous processes, i. e., between C5 and C6 for the C6 root, between C6 and C7 for the C7 nerve root, and between C7 and T1 for C8. A -

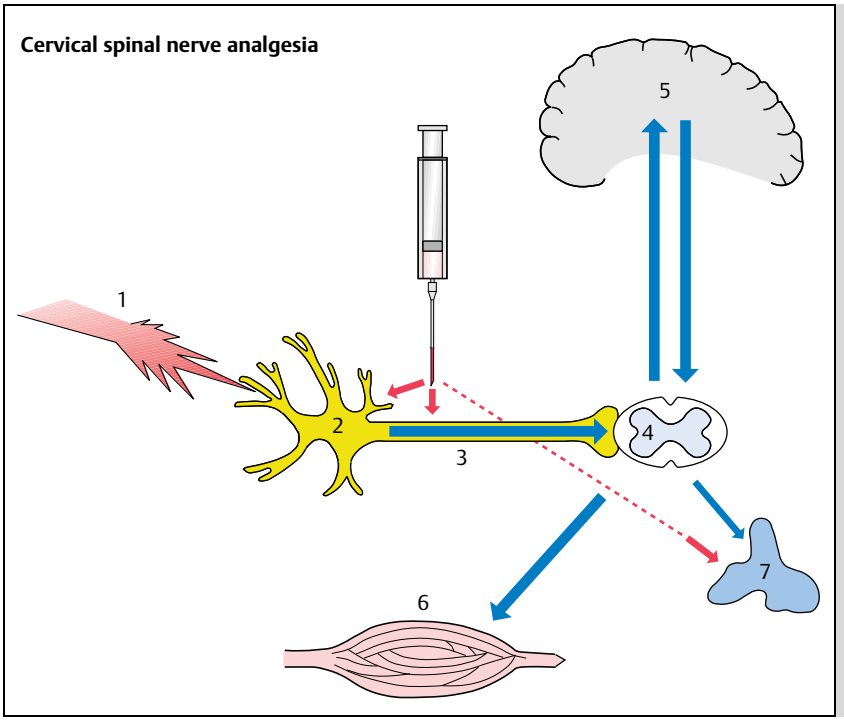


Fig. 7.6 CSPA. This is directed primarily toward the afferent fibers of the spinal nerve (3), but also targets the nociceptors found in the zygapophyseal joint capsule (2) and the muscles (6). The autonomic reaction (7) can be influenced by additionally infiltrating the cervical sympathetic nervous system.

stellate block can additionally be achieved at the last-mentioned segments. In our experience, this is more likely to be between C7 and T1 than between C6 and C7.

A cannula ~8 cm long, with a needle attached, is used. It is inserted vertical to the skin surface for 3–6 cm, until the tip of the needle reaches the flat overlapping mass located at the side of the cervical vertebral arch. With constant aspiration and light pre-injection of the local anesthetic, the tip of the needle is then moved in a superolateral direction around the lateral edge of the bone and further inserted ~1 cm. The rest of the local anesthetic is injected here. We use 10 mL of a 0.5–1.5 % lidocaine solution, but another type of local anesthetic can equally well be used. During the injection, the patient will first feel pain in the scapula, possibly followed by radiating spinal segmental pain in the arm. This pain phenomenon may not occur if the injection is conducted slowly with continual pre-injection of a fluid cushion (and aspiration). After the injection we keep the patient under observation in the waiting room for 30 minutes in case of possible complications and side effects. Patients are not fit to drive themselves on the day of the injection (**Figs. 7.7–7.28**).

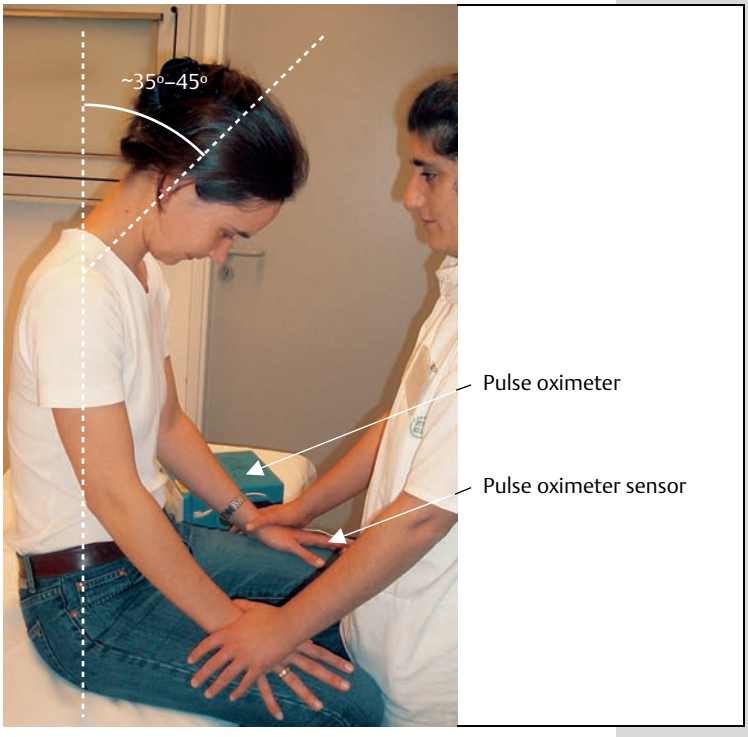
Effect of the Injection

Cervical spinal nerve analgesia serves predominantly to reach the ventral ramus of the spinal nerve at the point where it exits the intervertebral foramen in the lower cervical vertebral motor segments. The treatment targets the afferent fibers of the respective spinal nerve. The injection also reaches the dorsal ramus, the meningeal branch, and the sympathetic fibers of the rami communicantes. The posterior muscle groups, parts of the zygapophyseal joint capsule, and the corresponding ligamental connections are also infiltrated during the insertion of the needle. CSPA therefore has a complex local action, affecting pain originating in the cervical spine. This applies to acute pain that has just arisen as well as to chronic pain with increased motor and autonomic reactions.

Within a few minutes of the CSPA treatment, a warm feeling is felt in the arm and sometimes along the affected side of the head. The effect remains for a varying amount of time, ranging from 1–5 hours, depending on the local anesthetic's depot effect. Our investigations into the effectiveness of CSPA (Ripplinger 1977, Rubenthaler et al. 2000) have shown that a significant improvement in acute cervicobrachial syndromes is to be expected after only 1–3 injections, provided the affected segment can be accurately identified, clinically and neurologically, allowing treatment to be carried out in a precise manner.

NOTE

The aim of CSPA is not the complete analgesia and paralysis of spinal nerves, as is the case when preparing for surgery, but rather pain reduction and the desensitization of irritated neural structures in the cervical vertebral motor segment.

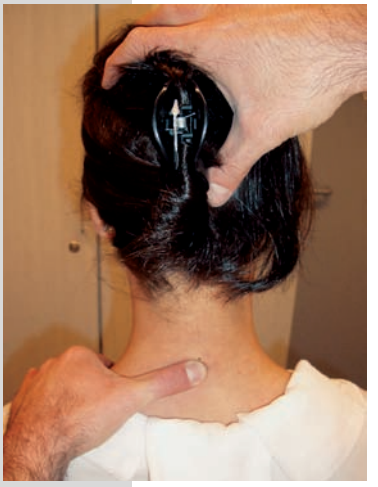


The CSPA Injection Procedure

Fig. 7.7 CPSA is conducted with the patient's cervical spine in flexion and their arms hanging down. The treating physician has to be able to look down on the shoulder/neck region. It is therefore best if tall patients sit on a chair or a stool. The flexed cervical spine makes it easier to palpate the spinous processes. The physician's assistant stands in front of the patient. Oxygen saturation and pulse frequency are monitored using a pulse oximeter. The treating physician or the assistant verbally monitors the patient throughout the procedure.

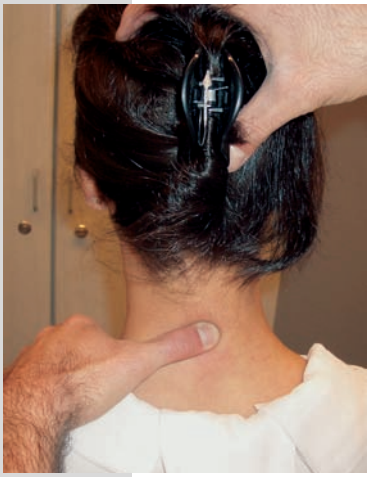
Fig. 7.8 The skin has to be intact and free from infection, especially in the injection area. Because of the need for repeated spraying, towels are placed over the upper parts of clothing.

Fig. 7.9 The hand position used when locating the C7 spinous process: When both hands are placed on the shoulders, the thumbs meet at C7.



a

b



c

Fig. 7.10a–c A further test is recommended at this stage to safely locate and palpate the tips of the C6 and C7 spinous processes. The left thumb is currently positioned over the C6 spinous process (**a**). The C6 spinous process can be palpated well during cervical flexion (**b**), but cannot be felt any more during cervical extension (**c**). In comparison, the position of the C7 spinous process remains palpable to the same extent during flexion and extension. The tips of the spinous processes can be reliably identified using these methods.

Fig. 7.11 Marking the palpated C6 and C7 spinous processes.

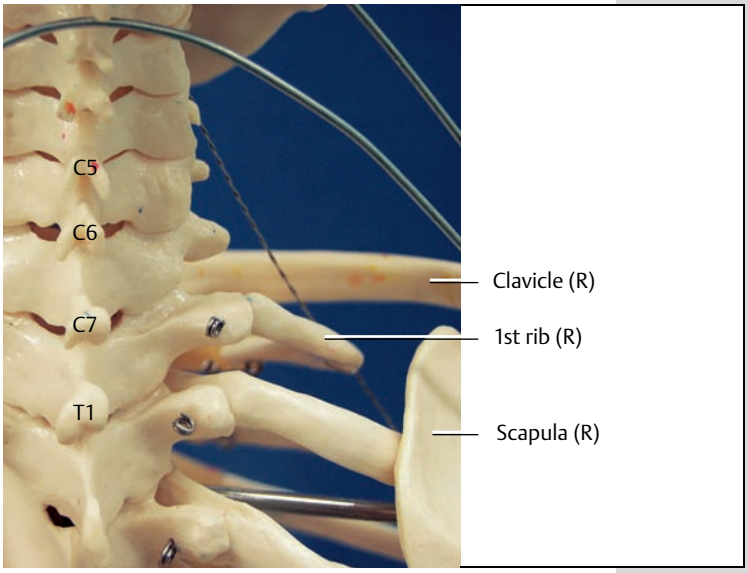


Fig. 7.12 Skeletal model: Cervical view from posterior.

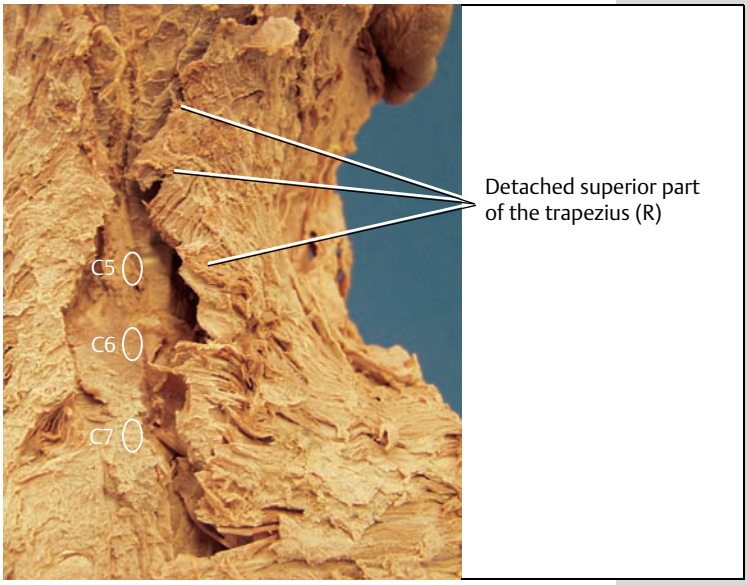


Fig. 7.13 Neck region from the posterior view shown on an anatomical specimen: The muscle attachments to the right and left of the spinous processes have been detached. The tips of the spinous processes can be found medially, directly under the skin. They can also be palpated well when the patient has well-developed muscles.

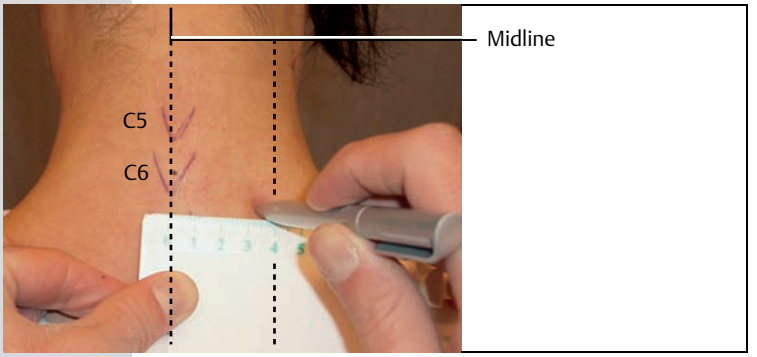


Fig. 7.14 Marking the injection site. The C5, C6, and C7 spinous processes are used for orientation. The injection site is found 3–4 cm lateral to the marked line and halfway between two spinous processes, i. e., between C5 and C6 for the C6 root, between C6 and C7 for the C7 nerve root, and between C7 and T1 for C8. A pen is used to mark the injection site by retracting the ink cartridge, placing the pen tip on the insertion point, and rotating. In this way the location of the site remains visible after disinfection.

Fig. 7.15 Skin disinfection using a colorless disinfectant spray.

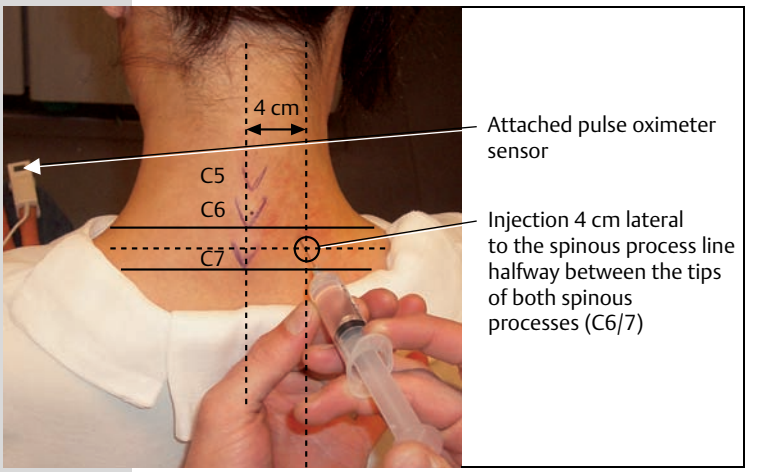


Fig. 7.16 Placing the needle on the marked injection site vertical to the surface of the skin.

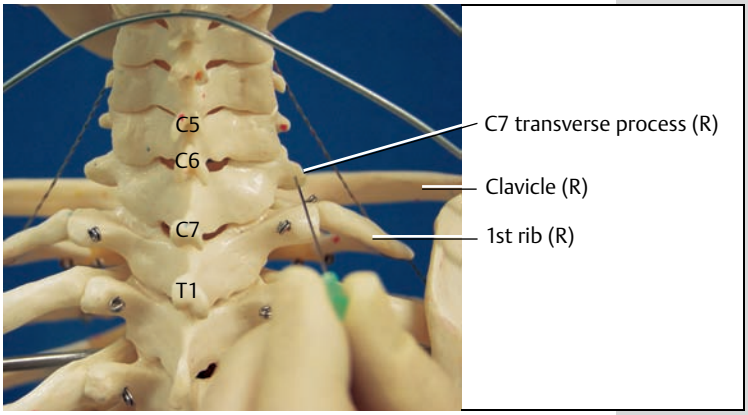


Fig. 7.17 Infiltration at C6/7. The injection site is located and directed toward the lateral edge of the cervical vertebral arch; in this case, toward the flat overlapping mass.

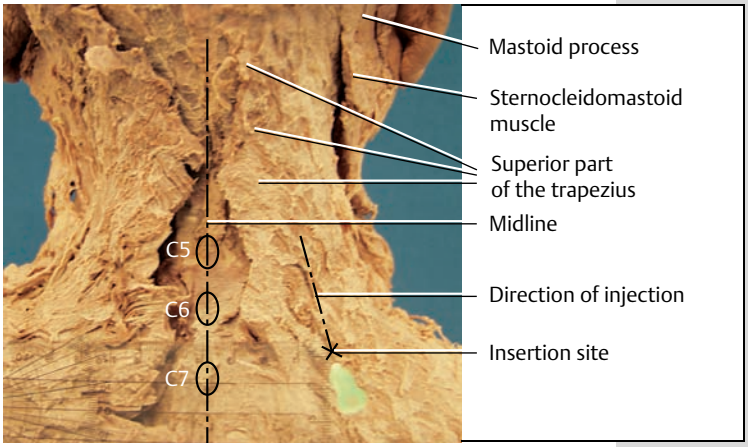


Fig. 7.18 Injection site demonstrated on an anatomical specimen: 3–4 cm to one side of the midline. Skin, superficial fatty tissue, and particularly thick muscle layers (muscle thickness depending on the patient's physique) have to be overcome before the needle comes in contact with the bone. The distance between skin and initial bone contact is 3–6 cm. For this reason a needle at least 8 cm long is required.

Fig. 7.19 Injection procedure, with slow insertion of the needle. The left hand maintains contact with the syringe while the back of the hand remains in contact with the patient. This ensures that the needle will follow the patient's movement if the patient suddenly moves backward.

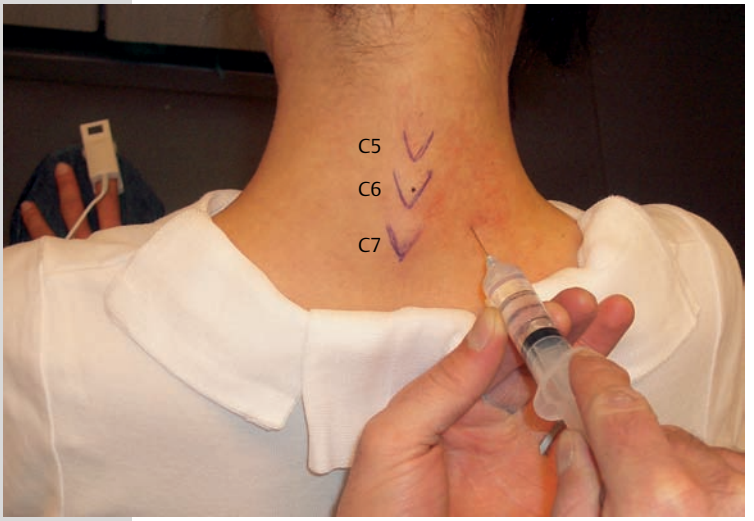


Fig. 7.20 When the skin resistance is overcome, the needle tip is inserted vertically, under continual aspiration and light pre-injection of local anesthetic, until contact is made with the bone.

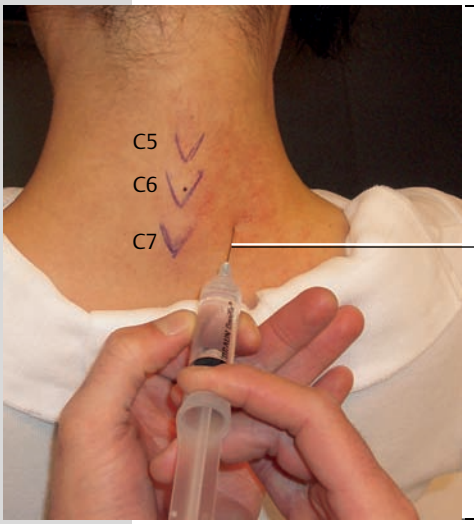


Fig. 7.21 A local anesthetic is pre-injected at bone contact to anesthetize the periosteum. The needle tip is moved superiorly and laterally until the edge of the bone can be felt. The needle is further inserted ~1 cm. The rest of the local anesthetic is injected here. A 10 mL syringe with a dilute local anesthetic (e. g., 0.5 % lidocaine) is therefore required. During the injection the patient will first complain of pain in the scapula, possibly followed up by radiating spinal segmental pain in the arm. This pain phenomenon may not be present if the injection is conducted slowly with continual pre-injection of a fluid cushion (and aspiration).

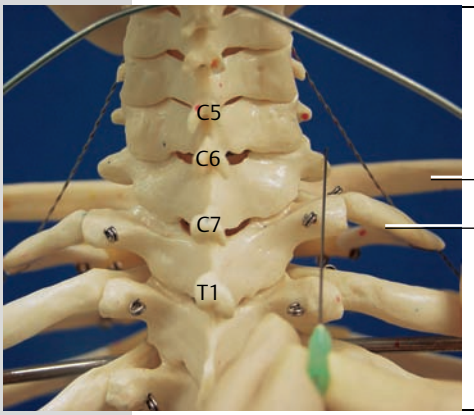


Fig. 7.22 Final position of the needle during cervical spinal nerve anesthesia, craniolateral to the lateral border of the vertebral arch.

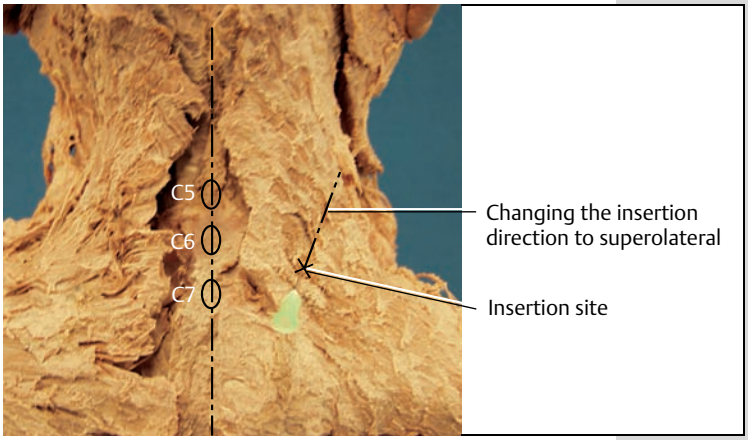


Fig. 7.23 Positioning of the needle on a specimen for a block between C6 and C7 (for the C7 nerve root).

Fig. 7.24 Final position of the needle at C6/7 during a C7 nerve root infiltration. Observe the handling of the syringe system using both hands, with the left hand resting on the patient.

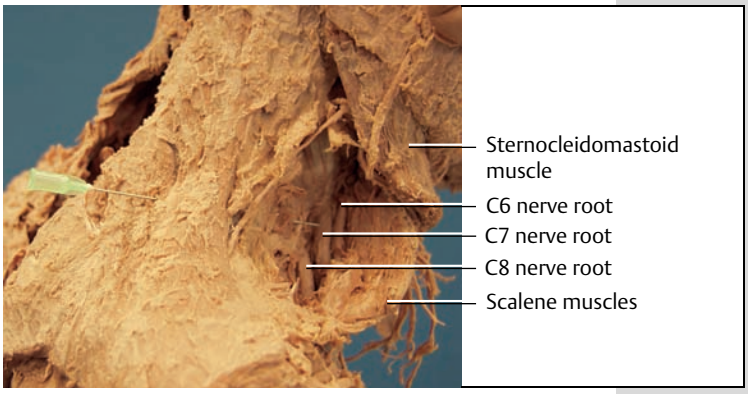


Fig. 7.25 The topography of the needle's position demonstrated on a specimen: The C6, C7, and C8 nerve roots can be seen laterally. The tip of the needle is located next to the C7 nerve root. The C6 and C8 nerve roots will also be infiltrated when 5 mL of local anesthetic is administered.

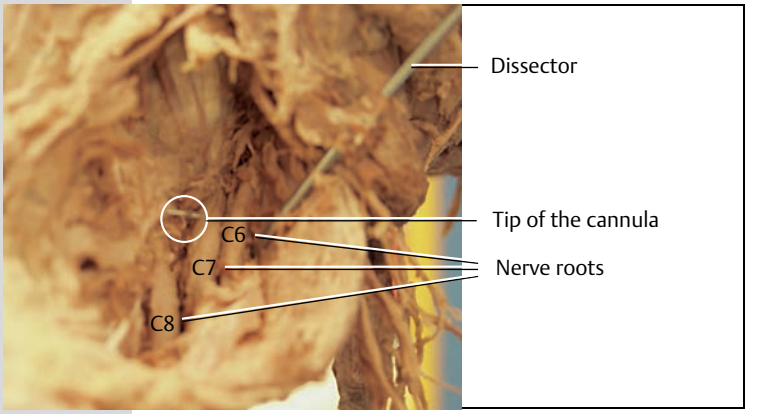


Fig. 7.26 The needle tip is pointing toward the C7 nerve root. The equally thick C6 and C8 nerve roots are located directly adjacent.

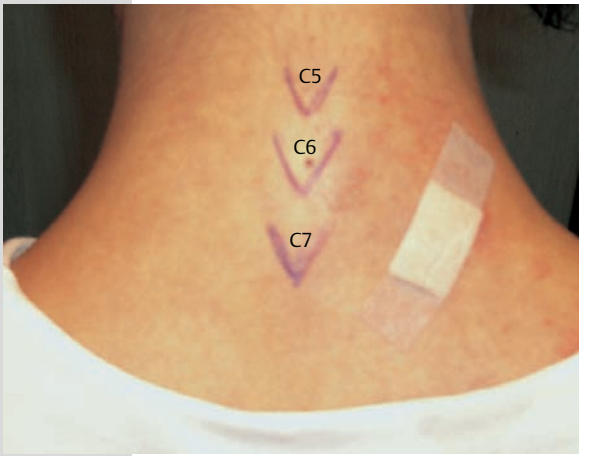


Fig. 7.27 Final photograph following C7 infiltration. A small nonallergic adhesive dressing is sufficient to cover the injection site. Because of the repeated injections planned at the same location, the dressing should be removed after an hour so as to avoid unnecessary skin reactions to it.

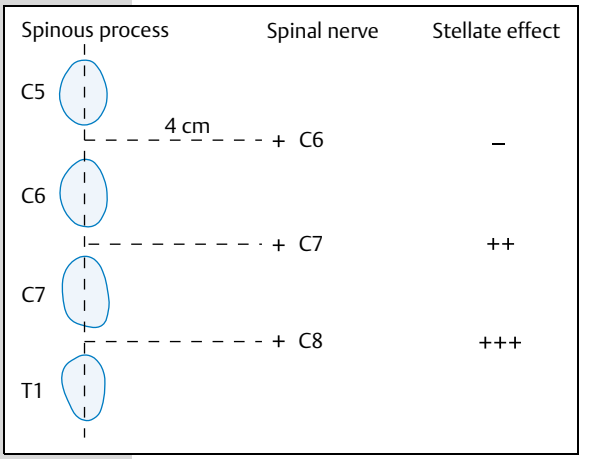


Fig. 7.28 Marking the injection site for CSPA from the posterior direction.

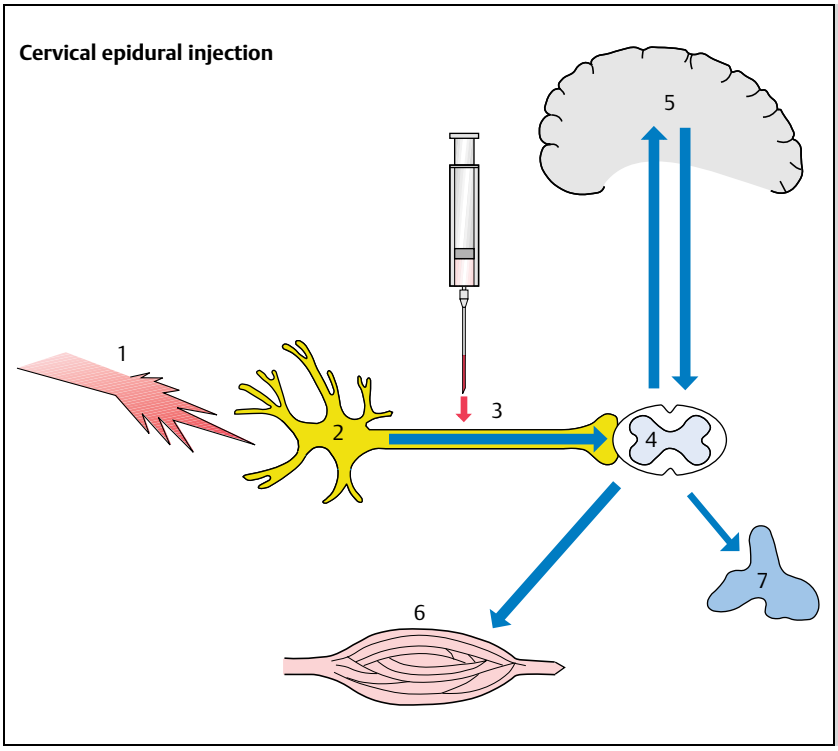


Fig. 7.29 The cervical epidural injection primarily targets the afferent fibers of the cervical spinal nerve root which have developed a nociceptor function following chronic irritation.

Cervical Epidural Injection

Principle

The injection of a steroid–saline solution into the epidural space in the lower cervical spine via the interlaminar space (**Fig. 7.29**).

This type of injection is used to treat chronic, mainly neuralgic pain in the lower cervical spine. The nerve root is flushed with anti-inflammatories at the site where it is mechanically irritated by osteophytes jutting out from the uncinate process and/or displaced intervertebral disk tissue where it is compressed by edematous swelling. This injection has two effects:

1 The injection causes a therapeutically induced reduction in local root inflammation. The space surrounding the spinal nerve is increased to such an extent that the blood flow through previously constricted epidural vein improves and the perineural edema can be reduced further.
2 The nociceptor function of the spinal nerve is reduced at the sensitive section of the nerve, at the point of direct mechanical compression.

Indication

Because of its relative complexity (see "Technique"), the cervical epidural injection is used only in cases of severe CBS, particularly if surgical decompression is being considered. This treatment method is only to be used as a last resort when all other methods have failed, because of the higher risks involved with the proximity to the CNS and the administration of epidural contrast agents.

The cervical epidural technique is contraindicated in the case of underlying neurological diseases, seizure disorders, known intolerance to contrast agents, skin infections at the injection site, and the usual corticosteroid contraindications.

Technique

The intervention is conducted with standby anesthesia, venous access, and EKG monitoring. The patient lies prone with a slightly flexed cervical spine so as to make interlaminary access easier. The cervical epidural space can best be reached between the arches of C5/C6, C6/C7, and C7/T1. The midline is ascertained using a needle and image-guided monitoring, and its position is marked on the skin. The height of the segment to be treated is ascertained using a lateral radiograph. A Spinocan 22 G cannula is then inserted until bony contact is made with the vertebral arch. This process is monitored by imaging. The needle, filled with a 0.9 % saline solution, is then inserted directly above the edge of the vertebral arch into the ligamentum flavum with constant pressure being applied to the plunger, similar to the posterior lumbar epidural injection. Sudden loss of resistance signals that the tip of the needle is positioned in the epidural space (spinolaminar line). The syringe barrel containing saline is then exchanged for one containing a contrast agent (we use Solutrast, as used in

myelography), which is connected to the Spinocan cannula via a connecting tube if necessary. This is followed by an epidurogram using 1–2 mL of contrast agent, verifying and documenting the epidural needle position. Finally, 5 mL of 0.9 % NaCl solution mixed with 10–20 mg of triamcinolone is injected.

The patient should lie on their painful side for 30 minutes after the injection so that the steroid solution can accumulate in the affected uncovertebral region (**Figs. 7.30–7.46**).

Effect of the Injection

In most cases, the cervical epidural injection with cortisone crystal suspension first relieves pain after several hours or during the following day. Some patients report an immediate effect. A treatment cycle consists of 1–2 epidural injections administered over 6–10 days. The epidural injections are complemented by daily CSPA.

Cervical Epidural Injection Procedure

Fig. 7.30 The intervention is conducted with venous access and EKG monitoring (and standby anesthesia, if necessary). The patient lies prone with a lightly flexed cervical spine to make the interlaminar access easier. Padding is placed under the thorax and abdomen so that they are slightly elevated. This enables the neck to be bent forward to allow the head to rest on further padding. The arms are placed parallel to the trunk and are not fixated. This enables an assistant to pull down the arms when necessary, so that the shoulders do not block the side view of the lower cervical segments.

Fig. 7.31 Optimal head/neck position for the cervical epidural injection. The patient's hair should be covered, with the hairline taped, before disinfection. A roentgen-transparent treatment table is required.

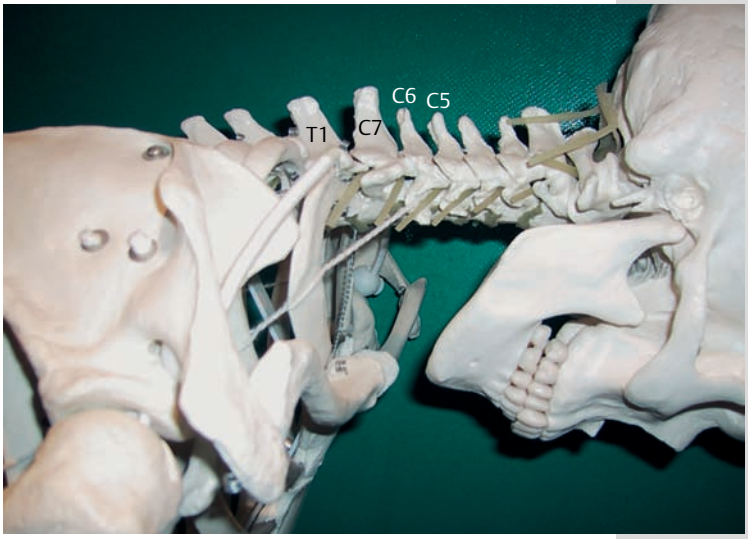


Fig. 7.32 The positioning for cervical epidural injection with neutralization of the cervical lordosis to enable interlaminar access, demonstrated on a skeleton.

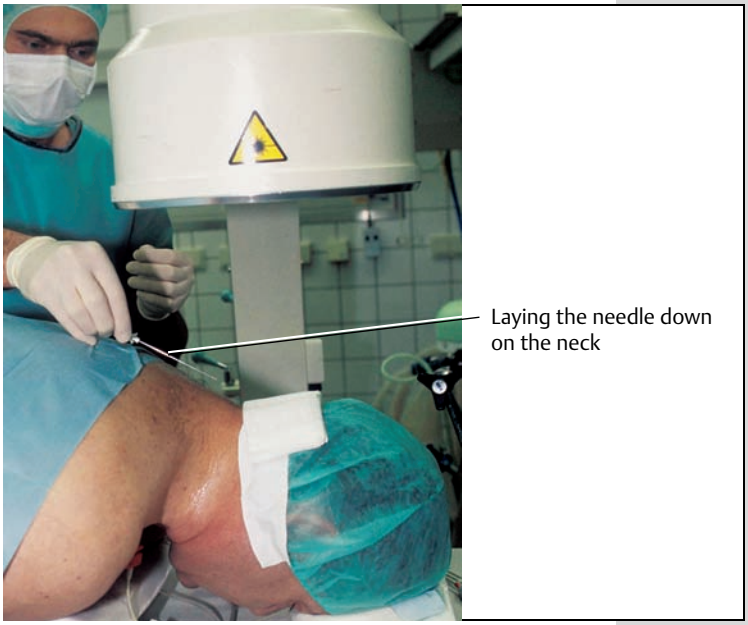


Fig. 7.33 The midline is located by laying a needle on the patient's neck under A/P fluoroscopy.

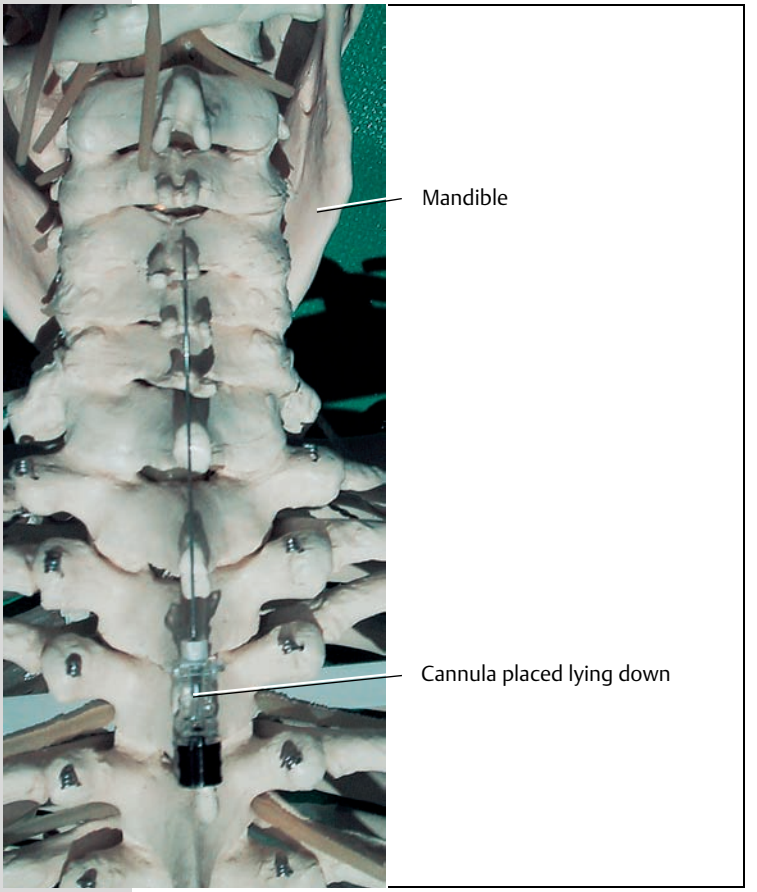


Fig. 7.34 A needle placed on a posterior skeletal cervical spine marks the midline. Directly adjacent to this is the optimal interlaminar point of entry to the posterior epidural space.

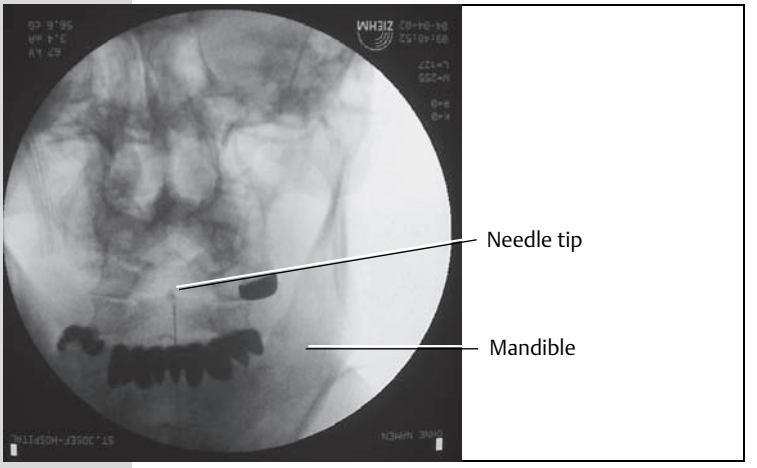


Fig. 7.35 Marking the midline in an A/P radiograph. The projection of the needle is often superimposed with the image of the viscerocranium. The midline is marked by rotating a pen tip on the insertion site (with the ink cartridge retracted). The location is marked in such a way that it remains visible following disinfection.

Fig. 7.36 The sterile part of the procedure begins by drawing the contrast agent into a syringe, a 0.9 % saline solution into another syringe, and a suspension of 0.9 % saline solution and 20 mg triamcinolone into a 5 mL syringe.

Fig. 7.37 Locating the height of the cannula between C5 and C6 using a lateral radiograph.

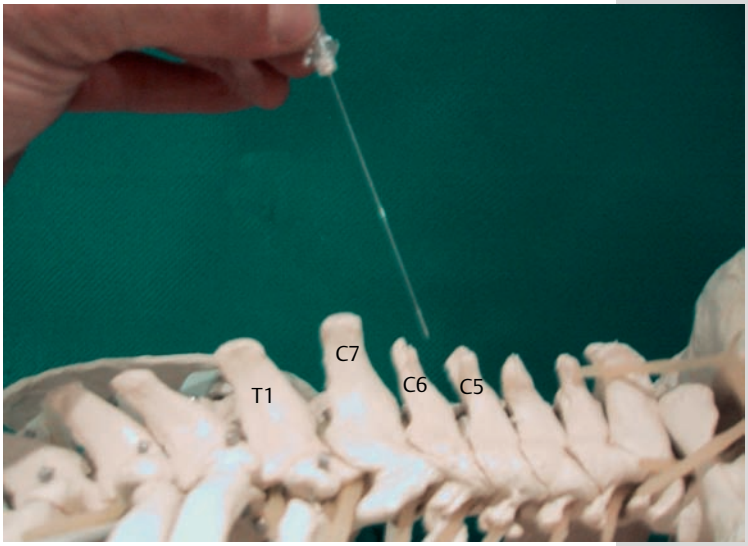


Fig. 7.38 The position of the Spinocan 22 G cannula between C5 and C6 as demonstrated on a skeletal model.

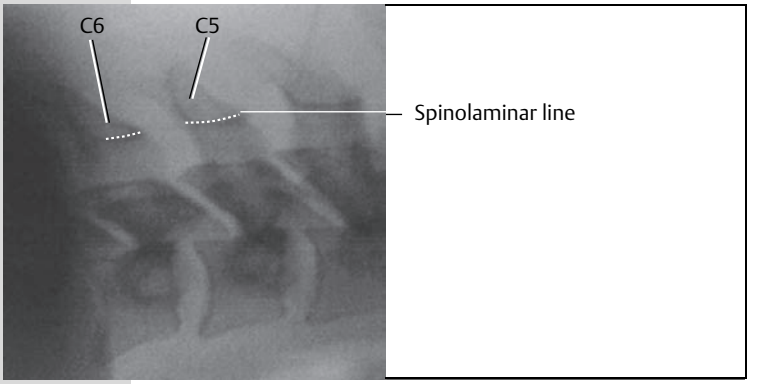
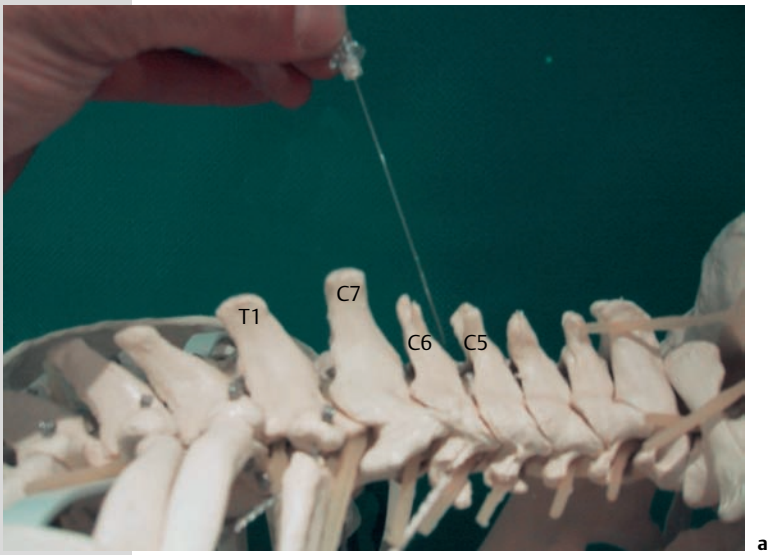
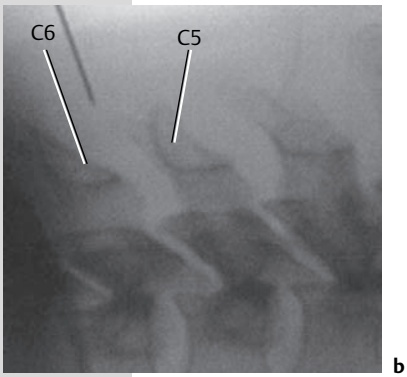


Fig. 7.39 Lateral imaging view of the cervical spine. If the lower segment is covered by the shoulders, an assistant should pull the arms down out of the way. The Spinocan 22 G cannula is inserted until bony contact is made with the vertebral arch. This process is monitored using imaging.



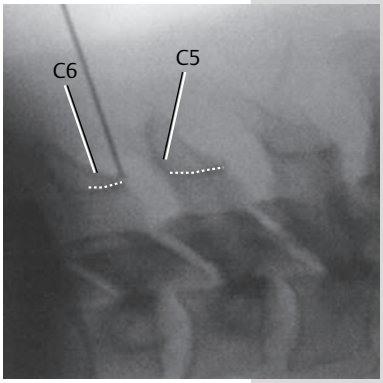
a



b

Fig. 7.40a, b The position of the needle during insertion, demonstrated first posterior to the lamina at C5/6 on a skeletal model (**a**) and during image conversion monitoring using a lateral radiograph (**b**).

a



b

Fig. 7.41a, b The position of the needle on the bony upper edge of the C6 lamina following further insertion of the needle, demonstrated on a skeletal model (**a**) and during monitoring with lateral radiography (**b**).

Fig. 7.42 Directly following the imaging above the C6 arch edge, the needle containing the saline solution is further inserted into the ligamentum flavum with constant pressure applied to the plunger, as for a posterior lumbar epidural injection. Sudden loss of resistance signals that the tip of the needle has reached an epidural position.

Fig. 7.43 Subsequently, the syringe barrel containg saline is replaced by the one containing the contrast agent (Solutrast, as used in myelography), which is connected to the Spinocan cannula via a connecting tube if necessary. Following aspiration, epidurography is conducted using 1–2 mL contrast agent. This should document the position of the needle and the epidural distribution of the contrast agent.

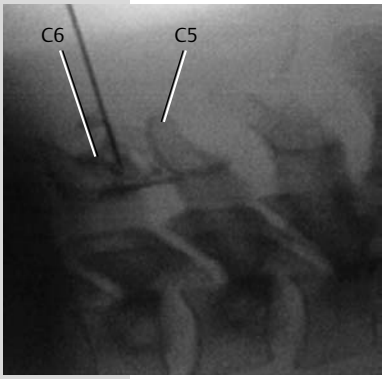


Fig. 7.44 The contrast agent is distributed linearly in the posterior epidural space (spinolaminar line), demonstrated here with an injection between C5 and C6.

Fig. 7.45 The syringe barrel containing the contrast agent is then replaced by the barrel containing the saline–steroid suspension. The back of the left hand is in contact with the patient, maintaining the correct needle position.

Fig. 7.46 This is followed by aspiration and then the injection of 5 mL of 0.9 % saline solution with 20 mg triamcinolone. The patient should lie on the painful side for 30 minutes after the injection so that the steroid solution can accumulate in the affected uncovertebral region.

Cervical Facet Infiltration (CxFI)

Principle

The switching off of nociceptors in the cervical zygapophyseal joint capsule using temporary blocks with a local anesthetic, and a steroid in addition if necessary.

Indication

CxFI is indicated in the cervical syndrome with pseudoradicular radiation into the arm, i. e., radiation that cannot be segmentally identified. Pain projected into the area between the scapulae, arising from the dorsal ramus of the spinal nerve, reacts well to this treatment. This type of pain increases when the neck is extended and axial compression of the zygapophyseal joints is present. Hyperlordotic neck pain is seen with the thoracic kyphosis associated with Scheuermann disease or osteoporosis.

Technique

The injection can be administered to patients in either a sitting or lying position. The neck should not be overly flexed, so that the interlaminar space remains largely closed. The posterior section of the zygapophyseal joints found at the back of the neck is reached by inserting a thin cannula ~2 cm paravertebral between the spinous processes of C5/6 and C6/7. These lordotic segments are the most common cause of pseudoradicular symptoms in the cervical area. The needle is inserted with permanent aspiration and pre-injection until bony contact is made with the posterior joint facet. This injection can also be carried out with sonographic monitoring (Grifka 1992).

At each location 2 mL of 0.5 % local anesthetic is injected into the zygapophyseal joint. A total of 10 mL of local anesthetic is therefore required. Initially, a total of 10 mg cortisone per 10 mL of a 0.5 % local anesthetic is recommended as the steroid component of the local anesthetic mixture (**Figs. 7.47–7.61**).

After the Injection

The procedure following CxFI is comparable to that following CSPA.

Cervical Facet Infiltration Procedure

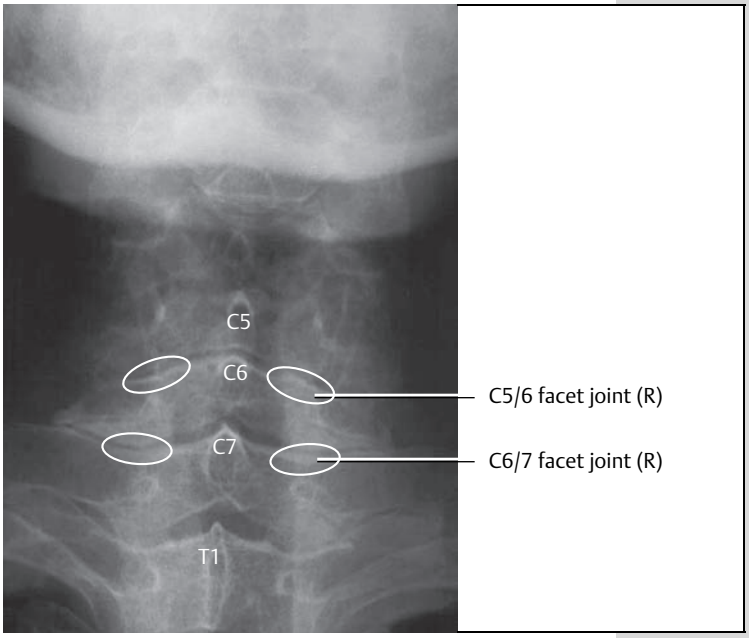


Fig. 7.47 An A/P radiograph of the inferior cervical section. The facet region and the posterior sections of the joint capsule in the inferior cervical vertebral motor segments are targeted.

Fig. 7.48 The patient sits during the injection. The cervical spine should not be placed in as much flexion (compared with the cervical epidural injection and CPSA). This ensures that the interlaminar area remains largely closed.

Fig. 7.49 An assistant positions the head. The skin must be intact and free of infection, especially in the injection area.

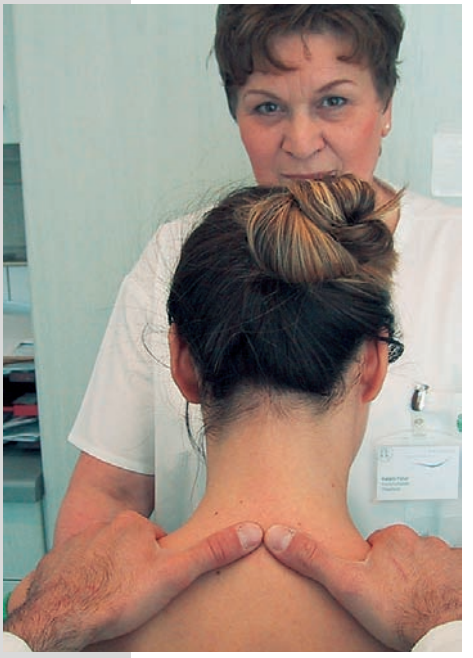


Fig. 7.50 The hand position used when locating the C7 spinous process: When both hands are placed on the shoulders, the thumbs meet at C7.

a b

Fig. 7.51a, b A further test is conducted at this stage to safely locate and palpate the tips of the C6 and C7 spinous processes. The left thumb is currently positioned over the C6 spinous process. The C6 spinous process can be palpated well during cervical flexion (**a**), but can no longer be felt during cervical extension (**b**). In comparison, the position of the C7 spinous process remains palpable to the same extent during flexion and extension. C6 and C7 are reliably identified using this method.

a

b

Fig. 7.52a, b Marking the spinous processes of C7 (**a**) and C6, C5 (**b**).

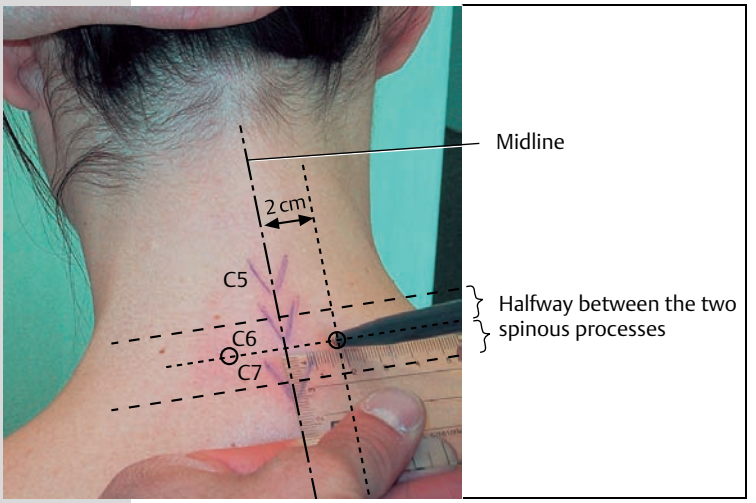


Fig. 7.53 The injection location for facet infiltration is marked 2 cm lateral of the midline between the spinous processes. The figure demonstrates marking for a bilateral infiltration between C6 and C7.

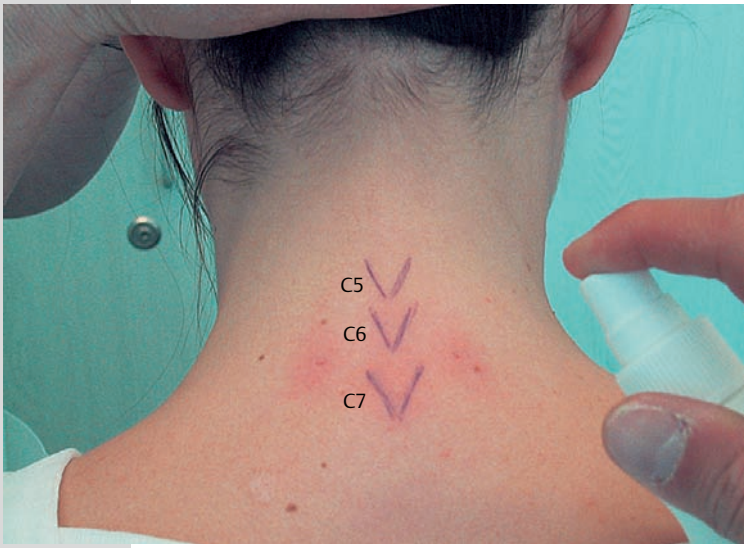


Fig. 7.54 Towels are used to protect clothing. The skin is then disinfected with a colorless disinfectant spray.

Fig. 7.55 The needle is inserted vertical to the skin surface.

Fig. 7.56 Both hands are used to guide the syringe. The left hand is resting on the patient.

Fig. 7.57 The needle is inserted until it comes in contact with the bone.

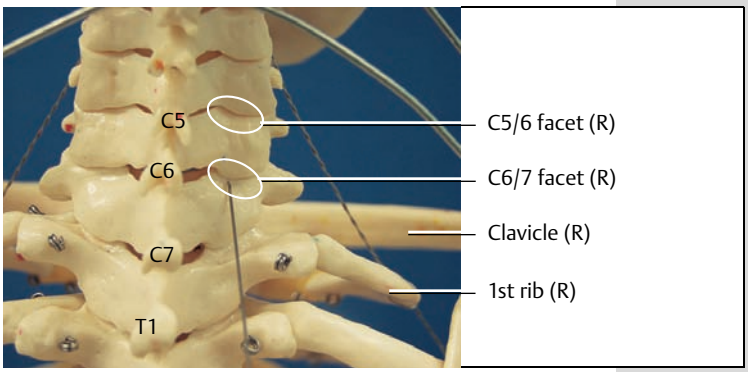


Fig. 7.58 Bony contact, as demonstrated on a skeleton. The bony contact is located more lateral. The needle tip can then be moved more medial. Further insertion is not necessary.

Fig. 7.59 Fan-shaped infiltration of the posterior zygapophyseal joint capsule at the level of bony contact. The syringe is guided by both hands.

Fig. 7.60 Facet infiltration: Posterior view.

Fig. 7.61 Facet infiltration: Lateral view.

Cervical Trigger Point Infiltration and Skin Infiltration

The therapeutic local anesthesia of regions not located in the immediate proximity of the cervical spine is used particularly in the treatment of secondary pain (see Chapter 1, "Mixed pain syndromes") in the shoulder/neck region. Favored areas include the muscle insertion sites for the trapezius, deltoids, and rhomboids. In cases of occipital neuralgia, the use of local injections into the upper edge of the scapulae with a lidocaine–cortisone crystal suspension mix is established practice. Painfully tense muscles can be relieved by combining stroking massage with infiltration of the shoulder/neck muscles using lidocaine or a similarly acting local anesthetic, particularly when injected into the upper trapezius border and the area of the rhomboids: 1–2 mL of weakly concentrated local anesthetic (0.5 % lidocaine, 0.125 % bupivacaine) is injected. It is advisable to administer a targeted infiltration into the cutaneous nerves in the occipital region at the same time (**Figs. 7.62–7.68**).

In combined injection treatment, infiltrations into muscle and muscle insertions are feasible alternatives to CSPA and cervical epidural injections, or can complement them.

Fig. 7.62 Trigger point infiltration at the right mastoid process, where the sternocleidomastoid muscle has its insertion. The skin in this area is innervated by the posterior ramus of the great auricular nerve and the end branches of the lesser occipital nerve.

Fig. 7.63 Trigger point infiltration at the superior nuchal line: A further area of insertion for the right sternocleidomastoid muscle, the site where the greater occipital nerve emerges (dorsal ramus of the second spinal nerve) in the subcutaneous layer, and in the direct vicinity of the lateral transverse path of the lesser occipital nerve coming from the cervical plexus.

Fig. 7.64 Trigger point infiltration at the exit point of the right third occipital nerve through the nuchal fascia. This nerve is one of the more thickly formed segmental posterior branches.

Fig. 7.65 The hand position used when conducting skin infiltration above the tense cervical paravertebral muscles. Both hands are used to guide the syringe.

Fig. 7.66 Conducting skin infiltrations in the right cervical paravertebral area.

Fig. 7.67 Completed skin infiltrations on the right with the distinct wheal formation.

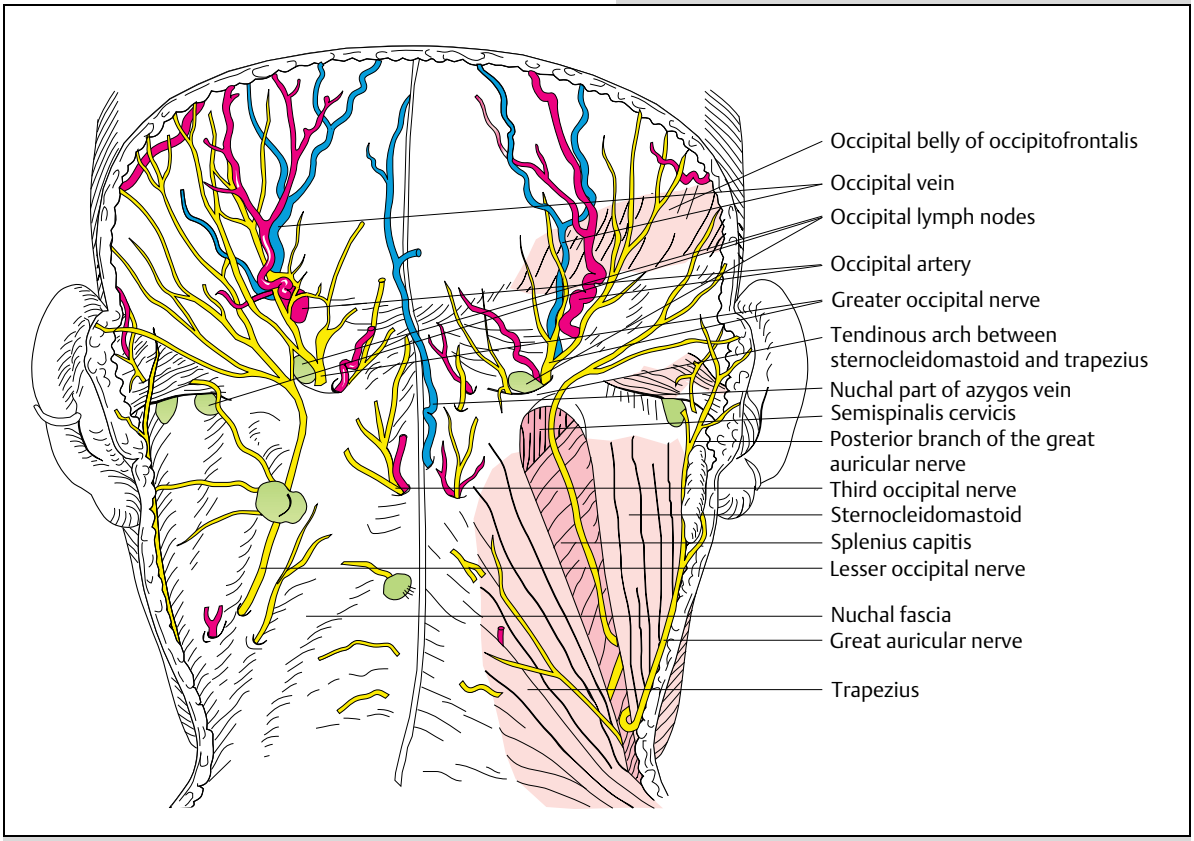


Fig. 7.68 Anatomical overview of the conductive pathways found in the occipital and nuchal regions. *Left*: Subcutaneous layer. *Right*: Subfascial layer.

8 Thoracic Injection Therapy

Acute and chronic pain in the thoracic spine plays only a minor role compared to that of the cervical and lumbar spine. This applies both to the incidence and to the severity of symptoms: Only 2% of all painful spinal syndromes affect the thoracic spine. In very rare cases, therapy-resistant nerve root irritation (intercostal neuralgia) occurs that requires surgical treatment.

Specialized Thoracic Neuroanatomy

The spinal canal is relatively narrow in the thoracic area and there is only a slender epidural space between the spinal cord, the bony surroundings, and the intervertebral disk. The canal is at its narrowest between T4 and T9.

The thoracic spine consists of the zygapophyseal joints, as well as the joints between the vertebrae and ribs (costovertebral joints, costotransverse joints), which jut out from the inferior part of the intervertebral foramen and push the spinal nerve out into the open section above (**Fig. 8.1**).

Because of the relatively large diameter of the intervertebral foramen, the osteogenic constrictions that are commonly found in the cervical spine, for example, are rarely seen in the thoracic spine. The intervertebral foramen is not adjacent to the intervertebral disk, as is the case in the cervical and lumbar spine. Rather, it is located at the same level as the vertebral body (**Fig. 8.2a, b**).

The displacement of the spinal cord segments in relation to the corresponding vertebral motor segments, as previously described for the cervical spine, continues in the thoracic spine. Between the 1st and 6th thoracic spinous process the displacement amounts to the height of two segments; between the 7th to 10th spinous processes, three segments. Ventral branches of the thoracic spinal nerve, the intercostal nerve, supply the wall of the rib cage, i.e., the intercostal muscles, the costotransverse joints, the parietal pleura, and the skin. A so-called intercostal neuralgia evolves when the thoracic spinal nerves are irritated.

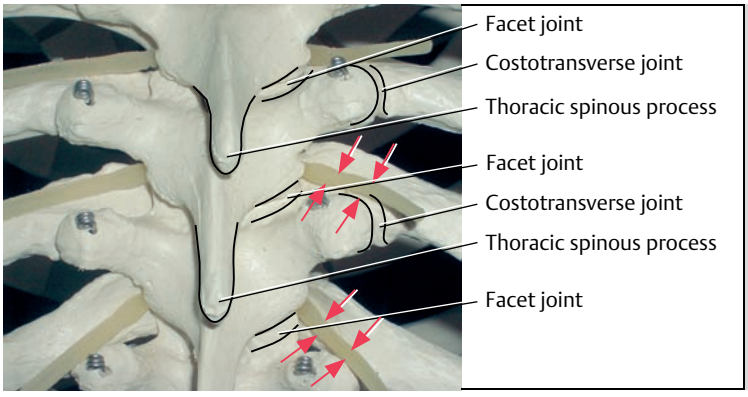
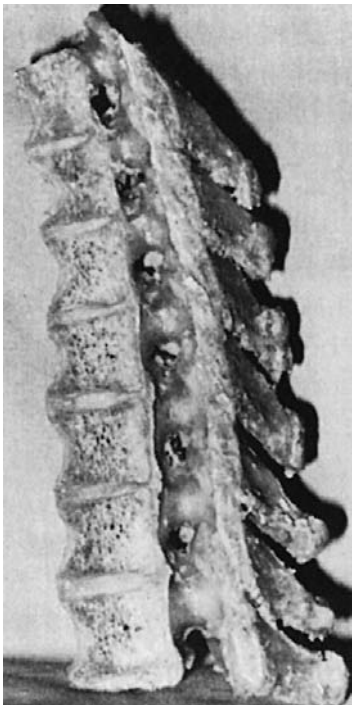
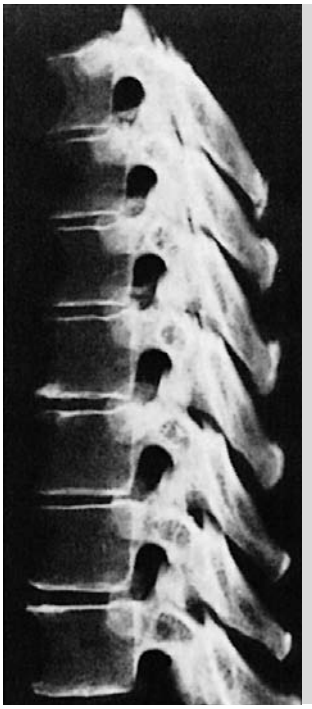


Fig. 8.1 Segments of the mid-thoracic spine on a skeleton, posterior view. In addition to the thoracic facet joints, the thoracic spine also contains costotransverse joints that jut out from the lower part of the intervertebral foramen and push the spinal nerve (arrows) into the freely open section above.



a



b

Fig. 8.2a, b Lateral sagittal section of the mid-thoracic spine shown on an anatomical specimen (**a**) and the radiological image of the same specimen (**b**) with a lateral view. The intervertebral foramina are not located next to the intervertebral disk, but rather at the level of the vertebral body.

Clinical Picture

The symptoms arising from a compromised thoracic spinal nerve root are characterized by a girdle-shaped pattern of pain with eventual discrete algesic disorders. Its topology is based on the dermatomal pattern. The boundaries between the individual dermatomes are not as well defined in the thoracic area as they are in the peripheral sections of the limbs. Another important diagnostic criterion for intercostal neuralgia due to degenerative changes is the dependency on position of the thoracic spine. Pain is relieved when the thoracic spine is unloaded or extended. Pain increases on loading and with certain rotary movements of the body. This provides information about how the condition should be treated.

Unloading the thoracic vertebral motor segments using the horizontal position is important in therapy. The application of all types of heat treatment is perceived as pleasant because the heat relieves the reflex hypertonicity of the trunk muscles, in particularly the paravertebral back extensors, and stimulates blood flow. These techniques are complemented by the use of manipulation, mainly in the form of traction, physiotherapy exercises to strengthen the trunk muscles, and local injections.

The irritation of thoracic spine nociceptive afferents is considerably more common than the irritation of spinal nerves. This form of irritation arises from incorrect posture or loading, or in some cases, segmental dysfunction or degenerative changes. It can cause extremely painful and treatment-refractory reflexive pain syndromes in the thoracic spine.

So-called viscerovertebral pain syndromes and pain syndromes associated with psychosocial problems can also manifest themselves in the thoracic spine.

Thoracic Injection Therapy

Thoracic Spinal Nerve Analgesia

Principle

Posterolateral injection of a local anesthetic (mixed with steroids when necessary) into the foraminoarticular region of the vertebral motor segment.

Indication

Any therapy-resistant intercostal neuralgia or intercostal neuralgia-like pain with girdle-shaped ipsilateral or bilateral radiation is an indication for thoracic spinal nerve analgesia (TSPA) treatment.

Technique

In order to carry out TSPA, bony contact has to be made with the transverse processes of the thoracic vertebral bodies. As the spinous process angle changes along the thoracic spine, the topographical relationship between spinous and transverse processes also varies. The spinous processes are found just under the transverse process belonging to the underlying segment between the 4th and 9th thoracic segments. Both of these corresponding points are found nearer to each other superior to T4 and inferior to T9 (Wolber 1999). The distance between the spinous process and the transverse process varies from 2.5 to 3.5 cm (**Fig. 8.3**).

A recent radiograph should be available for orientation purposes. The patient is positioned in a relaxed kyphotic posture with the arms hanging down by the sides. The infiltration can also be administered in a sitting position when a kyphosis table is not available.

T1–T4 Nerve Roots

Locate the lower edge of the spinous process to form a horizontal guideline. The transverse process line is found 3 cm lateral to this. A radiograph of the thoracic spine should be used for further orientation. The corresponding transverse process is found 2 cm superior to the horizontal guideline running from the lower edge of the spinous process. Insert a 6–10 cm needle vertically toward the corresponding transverse process. Retract the needle until the muscle fascia releases the needle tip. The injection is directed at a 20° caudal and 30–40° medial angle. When bony contact is lost, insert the needle a further 1–2 cm (approximately), then aspirate and inject ~1–2.5 mL per segment.

T5–T9 Nerve Roots

Locate the inferior edge of the T5, T6, T7, T8, or T9 spinous process. The transverse process line is found by running a line 3 cm horizontal and paraspinal to the lower edge of the spinous process. The corresponding transverse process can be palpated 3 cm superior to this line (the so-called "rule of threes"). The needle is inserted vertically to the skin, toward the transverse process. Following sufficient retraction of the needle out of the muscle fascia, the needle is then inserted at a 20° caudal and 30–40° medial angle. When bony contact is lost, insert the needle a further 1–2 cm (approximately), then aspirate and inject ~1–2.5 mL per segment.

It can be a problem for inexperienced practitioners to make targeted bony contact with the transverse process. In thoracic vertebral segments, the prominence of the transverse process is found significantly more posterior than the facet (**Fig. 8.4**). The administration of a thoracic facet infiltration (see "Thoracic Facet Infiltration" below) can therefore be used for orientation, to aid practitioners in ascertaining the insertion depth. In other words, the bony contact with the transverse process must be no deeper than the bony contact with the facet. A TSPA must not be administered without previous bony contact being made with the transverse process.

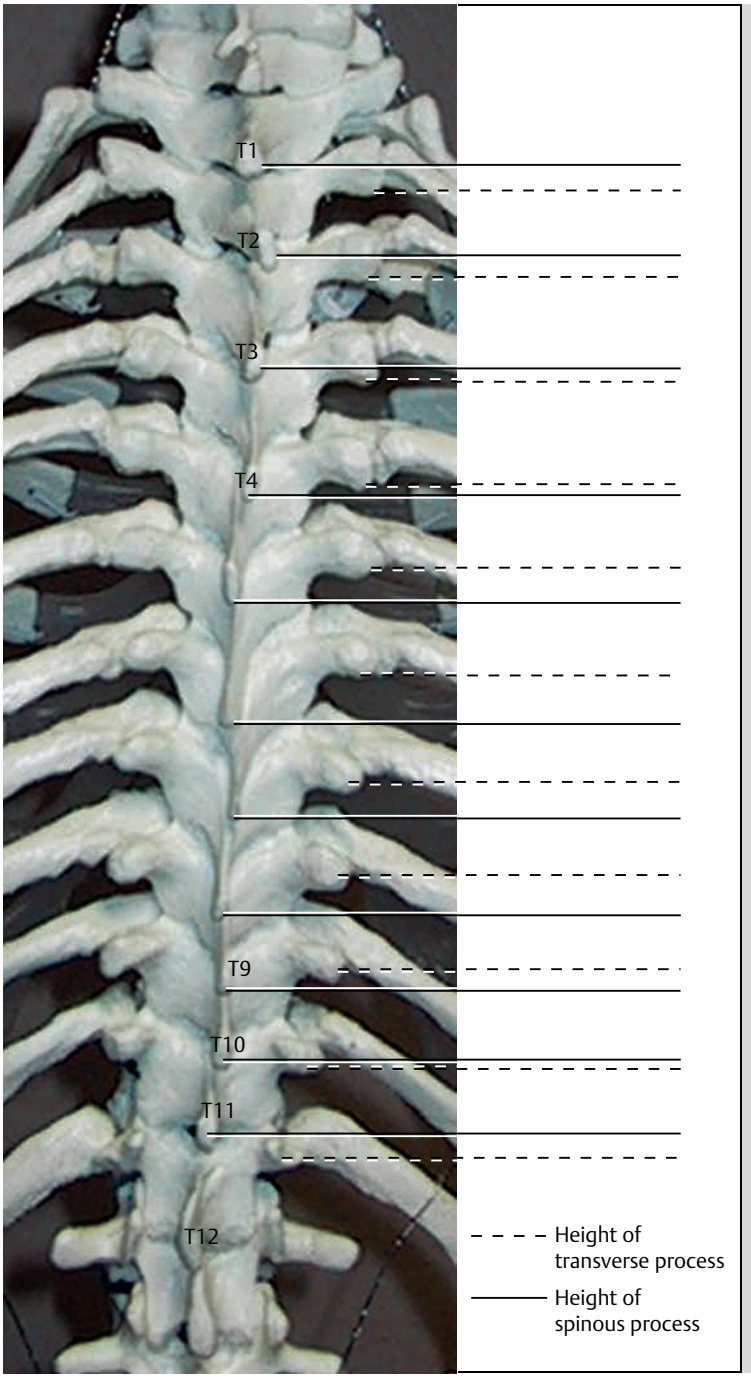


Fig. 8.3 Posterior view of the entire thoracic spine, shown on a skeleton. Topographic relationship between the spinous processes and the transverse processes: The spinous processes are found just under the transverse process belonging to the underlying segment between the 4th and 9th thoracic segments. Both of these corresponding points are found nearer to each other superior to T4 and inferior to T9.

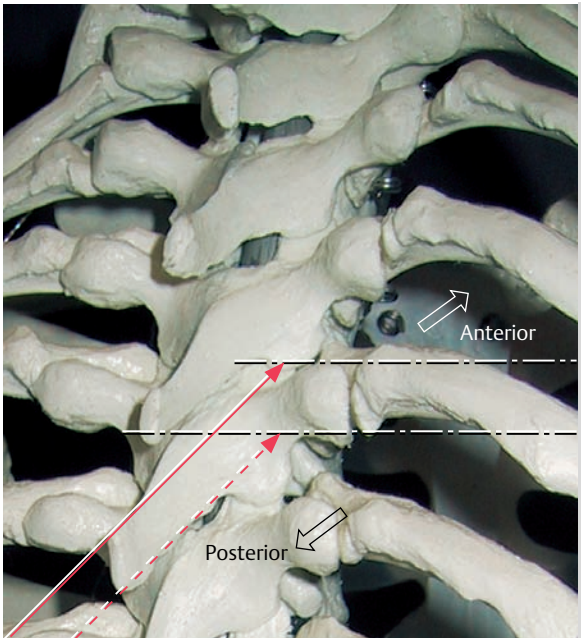


Fig. 8.4 The right posterior-lateral view of the upper thoracic spine shown on a skeleton. The prominence of the transverse processes is found significantly more posterior to the facet in the thoracic vertebral segments, i. e., during infiltration, the bony contact with the transverse process cannot be deeper than the bony contact with the facet.

Effects of Thoracic Spinal Nerve Analgesia

The patient may report segmental paresthesias. To reach a sufficient analgesic effect, 1–2 mL should be infiltrated per segment. An optimal block effect can be obtained even if the tip of the needle only comes within a few millimeters of the targeted site , because the anesthetic agent diffuses (**Figs. 8.5**–**8.16**).

Injection Procedure during Thoracic Spinal Nerve Analgesia

Fig. 8.5 TSPA is conducted with the patient sitting down and the cervical and thoracic spine in flexion. The patient's arms hang down by their sides. The flexed cervical spine makes it easier to palpate the vertebra prominens (C7 spinous process). The physician's assistant stands in front of the patient. Oxygen saturation and pulse frequency are monitored using a pulse oximeter. The treating physician or the assistant verbally monitors the patient throughout the procedure.

Fig. 8.6 The hand position used when locating the C7 spinous process. When both hands are placed on the shoulders, the thumbs meet at C7 (see also **Figs. 7.9–7.12**).

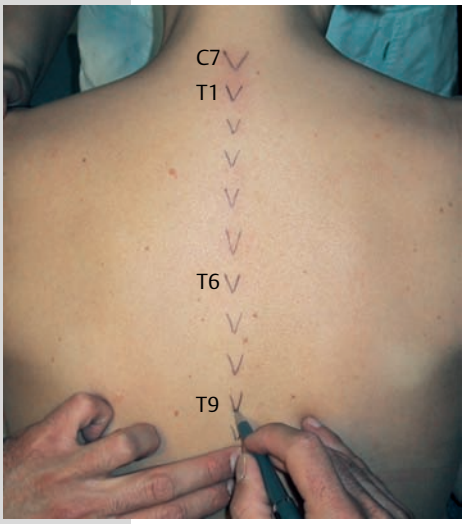


Fig. 8.7 The skin must be intact and free from infection, especially in the injection area. Palpating and marking the tips of the C7–T9 spinous processes.

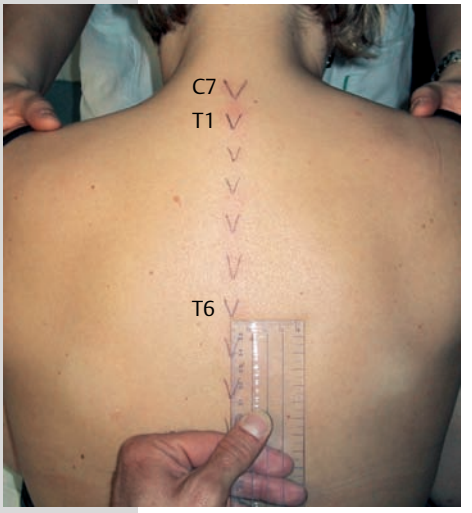


Fig. 8.8 Irritation point at the level of T6 on the right-hand side. Locating the lower edge of the T6 spinous process and the transverse process line to the right by placing a ruler horizontal to the inferior edge of T6.

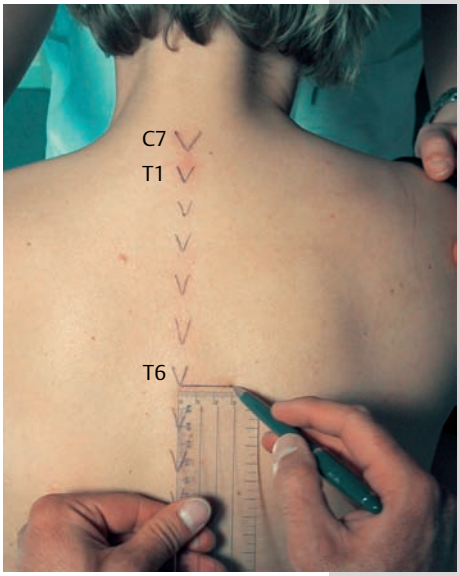


Fig. 8.9 The transverse process line is located 3 cm horizontal and paraspinal to the inferior edge of the T6 spinous process. Marking the distance 3 cm to the right.

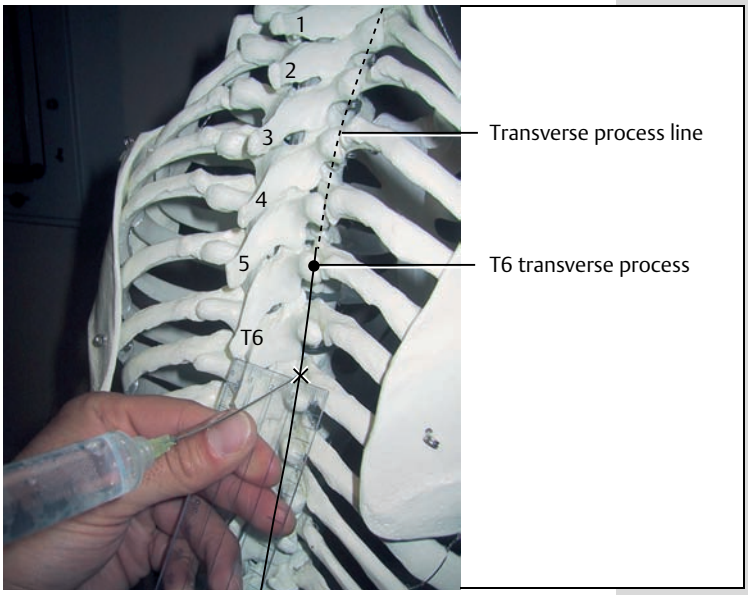


Fig. 8.10 Locating the transverse process line on a skeleton first of all 3 cm horizontal and paraspinal to the inferior edge of the T6 spinous process.

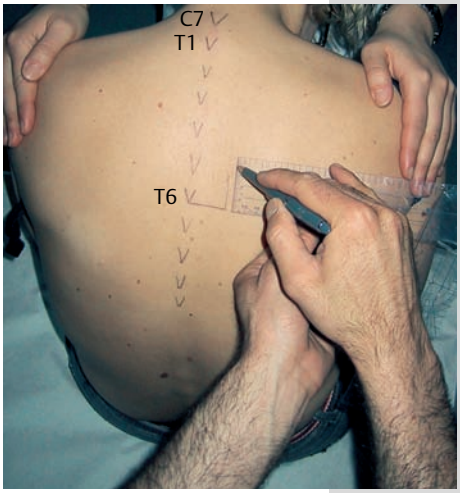


Fig. 8.11 The corresponding T6 transverse process is found parallel to the spinous process line 3 cm superior to the end of the 3 cm horizontal line (so-called "rule of threes"). Marking the 3 cm long vertical line. The injection site for the TSPA at T6 on the right-hand side is found at the end of this line. The injection site is marked by rotating a pen tip over the injection site (with retracted ink cartridge). The mark is still visible after disinfection.

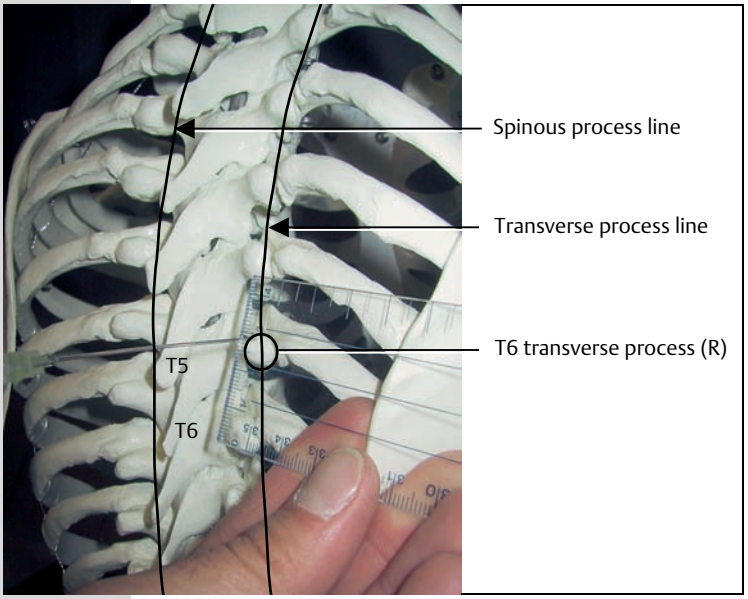


Fig. 8.12 The position of the needle over the right T6 transverse process, shown on a skeleton.

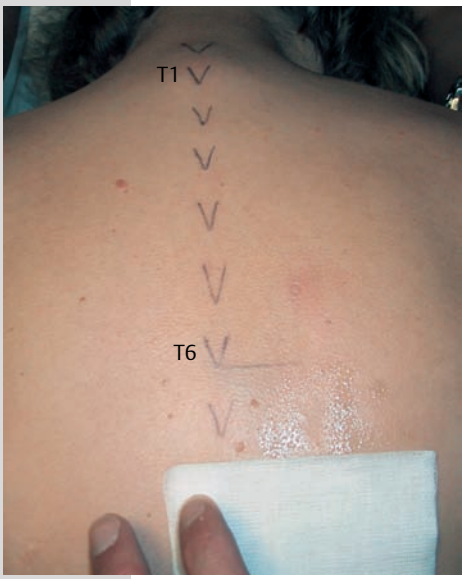


Fig. 8.13 Compresses are placed caudal to the injection site. The skin is then disinfected with a colorless disinfectant spray.

Fig. 8.14 To begin with, the needle is inserted vertical, directed toward the transverse process. The handling of the syringe with the slow insertion of the cannula. The left hand maintains contact with the syringe while the back of the hand remains in contact with the patient. This ensures that the syringe will follow the patient's movement if they move backward suddenly.

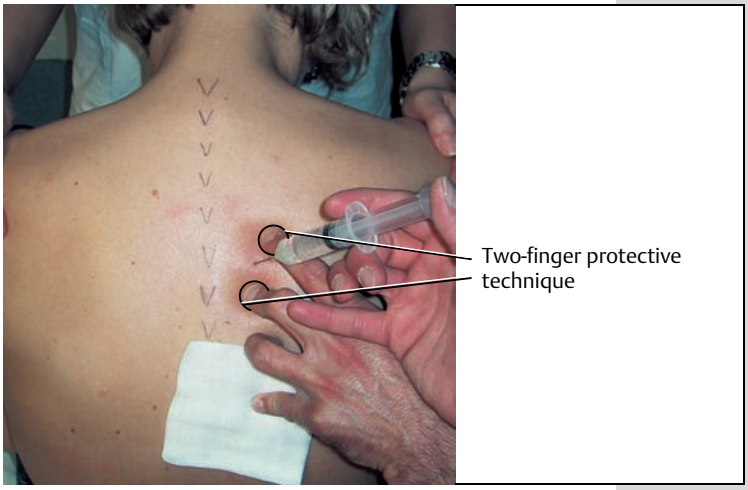


Fig. 8.15 Protecting the T5/6 and T6/7 intercostal spaces using the two-finger technique. To reach the spinal nerve the cannula has first to be retracted sufficiently. It is then inserted a further ~1–2 cm at an angle of 10–20° in a caudal direction and 30–40° in a medial direction, bypassing the transverse process. After aspiration, ~1–2.5 mL is injected.

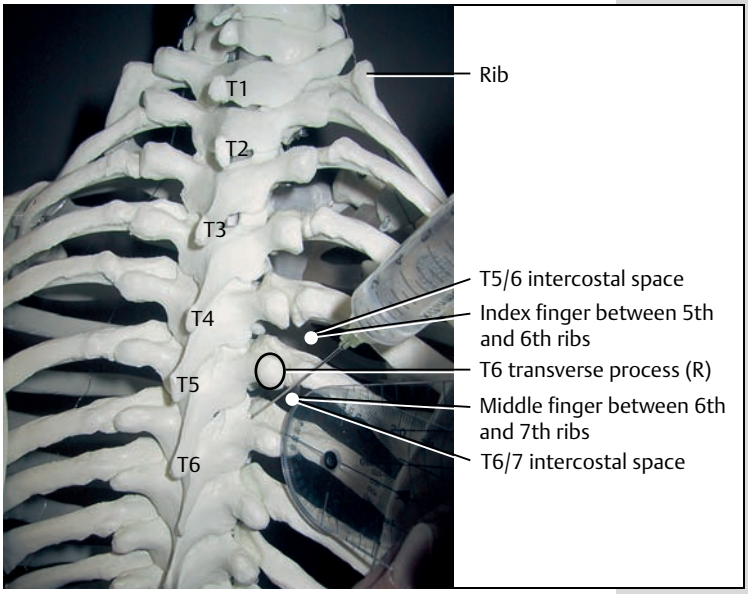


Fig. 8.16 Final position of the needle during a right TSPA at T6 as demonstrated on a skeleton.

Thoracic Facet Infiltration (TxFI)

Principle

The switching off of nociceptors in the thoracic zygapophyseal joint capsule using temporary blocks with a local anesthetic and an additional steroid when necessary.

Indication

Symptoms that originate in the zygapophyseal joints, i. e., thoracic facet syndromes, tense thoracic paravertebral muscles without sensomotoric deficits, pseudoradicular thoracic syndromes.

Technique

The patient either sits with a kyphotic thoracic spine or lies on a treatment table which allows a kyphotic position. The injection site is found ~1 cm paraspinal to the upper edge of the spinous process. This paraspinal distance should not exceed 1.5 cm. The use of a radiograph is absolutely necessary for orientation. The zygapophyseal joint capsule is reached with the vertical insertion of a 6–8 cm cannula until bone/capsule contact is made. In slim patients the facet may be reached after 3–4 cm, but in obese patients the needle must be at least 8 cm long. When the tip of the cannula intrudes into the joint or the joint capsule, patients will experience their typical radiating pain. Intraarticular positioning of the needle is not required. Generally speaking, a periarticular–pericapsular infiltration is more than sufficient. Following aspiration, 1–2 mL is infiltrated into each segment (**Figs. 8.17–8.24**).

Thoracic Facet Infiltration Procedures

Fig. 8.17 The thoracic facet infiltration is conducted with the patient sitting down and the cervical and thoracic spine in flexion. The patient's arms hang down by their side. The flexed cervical spine makes it easier to palpate the vertebra prominens (C7 spinous process). Oxygen saturation and pulse frequency are monitored using a pulse oximeter. The patient should be constantly monitored verbally.

Fig. 8.18 The skin has to be intact and free from infection, especially in the injection area.

Fig. 8.19 Locating and marking the C7 spinous process.

Fig. 8.20 Palpating and marking the tips of the T1–T5 spinous processes.

Fig. 8.21 The injection site is found ~1–1.5 cm paraspinal to the upper edge of the spinous process. This paraspinal distance should not exceed 1.5 cm. The use of a radiograph is absolutely necessary for orientation. The injection site is marked with a pen (with retracted ink cartridge).

Fig. 8.22 After disinfection, a 6–8 cm cannula is inserted vertically at the marked injection site. Both hands are used to guide the injection system. The left hand rests on the patient.

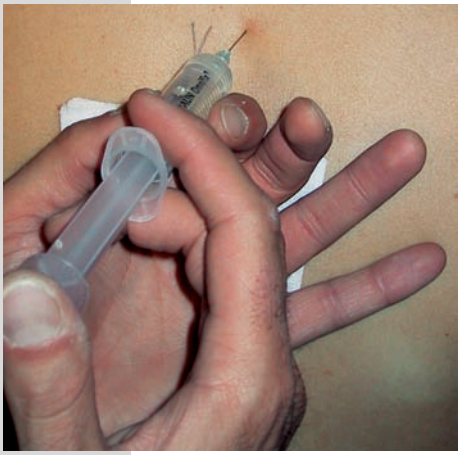


Fig. 8.23 The needle is inserted until it comes in contact with the bone and capsule. The facet may be reached after only 3–4 cm in slim patients, but for obese patients the needle must be at least 8 cm long. Using both hands, ~1–2 mL of local anesthetic is infiltrated in fan-shaped into the **posterior capsule** of the zygapophyseal joint when contact is made with the bone and following aspiration.

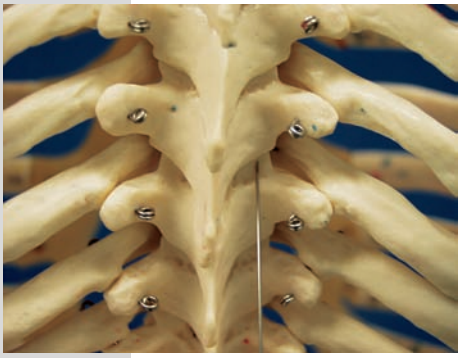


Fig. 8.24 The position of the needle in thoracic facet infiltration shown on a skeleton. The facet complex is found 1–1.5 cm lateral to the midline at the same level as the upper edge of the spinous process.

Costotransverse Block (CTB)

Principle

The switching off of nociceptors in the costotransverse joint capsule using temporary blocks with a local anesthetic and an additional steroid when necessary.

Technique

All ribs, except for the 11th and 12th ribs, are additionally articulated to the vertebrae at the transverse process (costotransverse joints).

Palpation of the costovertebral joint or the associated area of irritation (insertion of levator costae) is required before infiltrating the costotransverse joint. It is possible to examine the nociceptive reaction in the costotransverse joints using diagnostic manual therapy methods. Areas of irritation are most likely to be observed between T5 and T9.

The costotransverse joints are angled at 40–60°, starting from the spinous process plane. For this reason, the chosen injection site must be located sufficiently laterally. A radiograph is required for orientation.

When patients are placed in a kyphotic position, the injection site is found 3 cm lateral to the spinous process line (= transverse process line). The injection puncture site is determined using the "rule of threes" (see "Thoracic Spinal Nerve Analgesia", above). The intercostal space should be protected using the two-finger technique, to prevent an intrapleural needle position. A needle length of 5–8 cm is chosen, depending on the patient's anatomic condition. The needle is inserted medially, at an angle of 45–60°, until contact is made with the capsule. Following aspiration, it is recommended that 1–2 mL (no more) of local anesthetic be infiltrated. This can be mixed with steroids if necessary. The nociceptive input can also be blocked with a pericapsular needle position by administering in infiltration of up to 2 mL (**Figs. 8.25, 8.26**).

Fig. 8.25 Protecting the intercostal space with the aid of the two-finger technique during the costotransverse block (CTB), demonstrated on a skeleton.

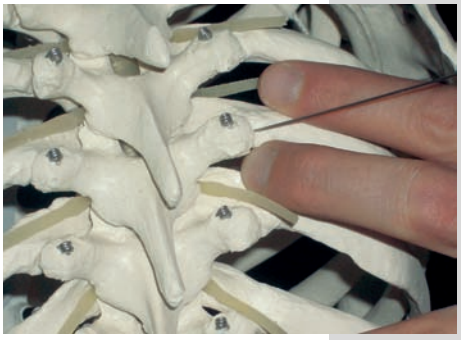


Fig. 8.26 The position of the needle during the CTB, shown on a skeleton.

9 Lumbar Injection Therapy

Pain in the lumbar spine primarily arises in the two inferior lumbar vertebral motor segments, i. e., L4/L5 and L5/S1. One reason why the most marked disorders of form and function are found here is because of the exceptional loading placed on the inferior lumbar spine. Another reason is that spinal nerves and their efferent branches are found in the immediate vicinity. The sacroiliac joints also usually contribute to this painful situation. They belong functionally to the inferior lumbar vertebral motor segments, and the dorsal ramus of the spinal nerve connects them with the S1 nerve root. The special topographical relationship between the musculoskeletal system and the nervous system in this region is of particular importance not only to pain therapists, but also to surgeons.

Specialized Lumbar Neuroanatomy

The vertebral body and the intervertebral disk form the anterior border of the lumbar spinal canal, the ligamentum flavum and the vertebral arch the posterior border. The pedicles and the intervertebral foramina are found laterally. The lumbar vertebral canal is a cylindrical cavity that changes its shape and volume with every movement of the trunk: Trunk flexion increases its volume, and trunk extension causes it to narrow.

The contents of the lumbar vertebral canal include the dural sac, nerve roots, and peridural tissue. This consists of veins and fatty tissue and encases the nerve root, ensuring that it is protected from the bony borders of the vertebral canal even during extreme movements of the lumbar spine.

The displacement of spinal cord segments in relation to their corresponding motor segment in the vertebral column is at its most extreme in the lumbar spine. The inferior end of the spinal cord extends only to the first or second lumbar vertebra. The spinal nerves exit the vertebral canal through their corresponding intervertebral foramina inferior to this, and travel for a long distance in the subarachnoid space, where they are usually found laterally. There is thus no need to worry about nerve injuries when conducting medial lumbar punctures and myelography, or during transdural disk punctures.

Paramedian protrusions and prolapses can cause the intrathecal compression of spinal nerves and more deeply situated nerve root syndromes. The entire length of the long inferior spinal nerves and the filum terminale, the final fibers of the spinal cord that extend down to the second coccygeal vertebra, is called the cauda equina.

After leaving the dural sac, the nerve root travels in a direction dictated by the segmental level. The more inferior the nerve root, the more acutely angled it is as it exits the dural sac. This explains why different topographical relationships are observable between each nerve root and intervertebral disk in the lumbar vertebral motor segments. The exit point for the L4 nerve root is found at the same level as the L3 vertebral body, the L5 nerve root exits the dural sac at the height of the inferior edge of the L4 vertebral body, and the S1 nerve root at the inferior edge of the L5 vertebral body (**Fig. 9.1**).

A L4/5 intervertebral disk prolapse (**Fig. 9.1**, arrows) exerts pressure primarily on the L5 nerve root. The L4 nerve root is affected only when the prolapse is very large and is displaced in a lateral or superior direction, as a result of the L4 nerve root passing superior to the L4/5 intervertebral disk. The S1 and L5 nerve roots can be affected at the same time at the L5/S1 intervertebral segment, even by a small lateral prolapse (**Fig. 9.1**, arrows). The L5 spinal nerve root lies in the upper section of the intervertebral foramen, directly on the outer lamellae of the intervertebral disk. The L5 nerve root has only a very small amount of free space within the L5/S1 intervertebral foramen. The lumbar nerve roots are affected by intervertebral disks only in the two inferior segments: It is here that there is the most danger of compression arising from an intervertebral disk.

The foraminal articular region, with the spinal nerves exiting the intervertebral foramen and the efferent nerve branches, is of particular interest. Directly after its exit from the intervertebral foramen, the spinal nerve divides into a thicker ventral ramus, a somewhat thinner dorsal ramus, and a tiny sinuvertebral nerve. The dorsal ramus divides into a lateral branch that runs to the facets and a medial branch that runs to the spinous process (**Fig. 9.2**). The recurrent branch (meningeal branch, sinuvertebral nerve, see **Fig. 9.3**) passes through the intervertebral foramen, back into the spinal canal, and innervates the rear section of the anulus fibrosus, the posterior longitudinal ligament, and the dura.

Figure 9.3 (second spinal nerve) demonstrates how close the spinal nerves and their branches are to the muscles and joints, and also to the sympathetic trunk via the ramus communicans connection in the foraminal ar-

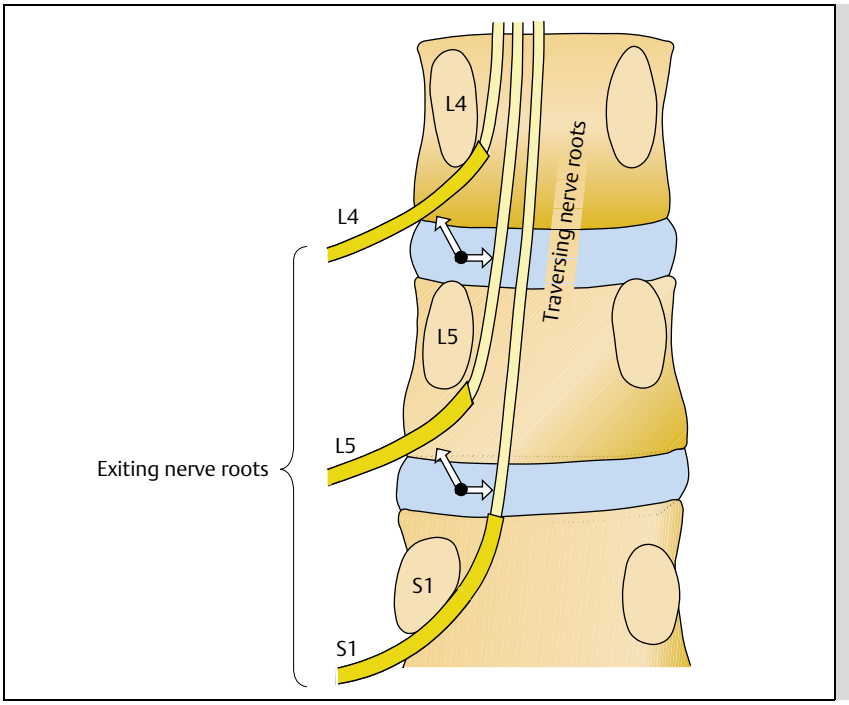


Fig. 9.1 Nerve roots that traverse and exit in the inferior lumbar vertebral canal. The traversing nerve roots run vertically in the vertebral canal, intrathecally and extrathecally, and pass over the intervertebral disk. The spinal nerve roots lateral to the medial pedicle border are labeled as exiting nerve roots and have an oblique to horizontal course. The S1 nerve root is a traversing nerve root up to its exit point from the sacral canal.

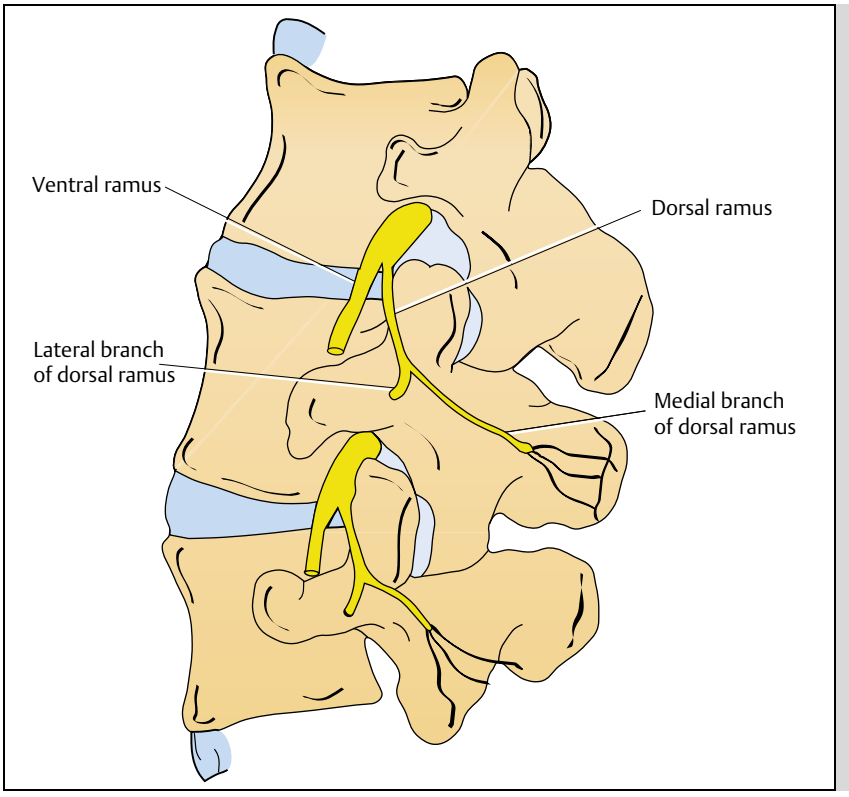


Fig. 9.2 Lumbar spinal nerve with ventral ramus and dorsal ramus (according to: Bogduk 1997).

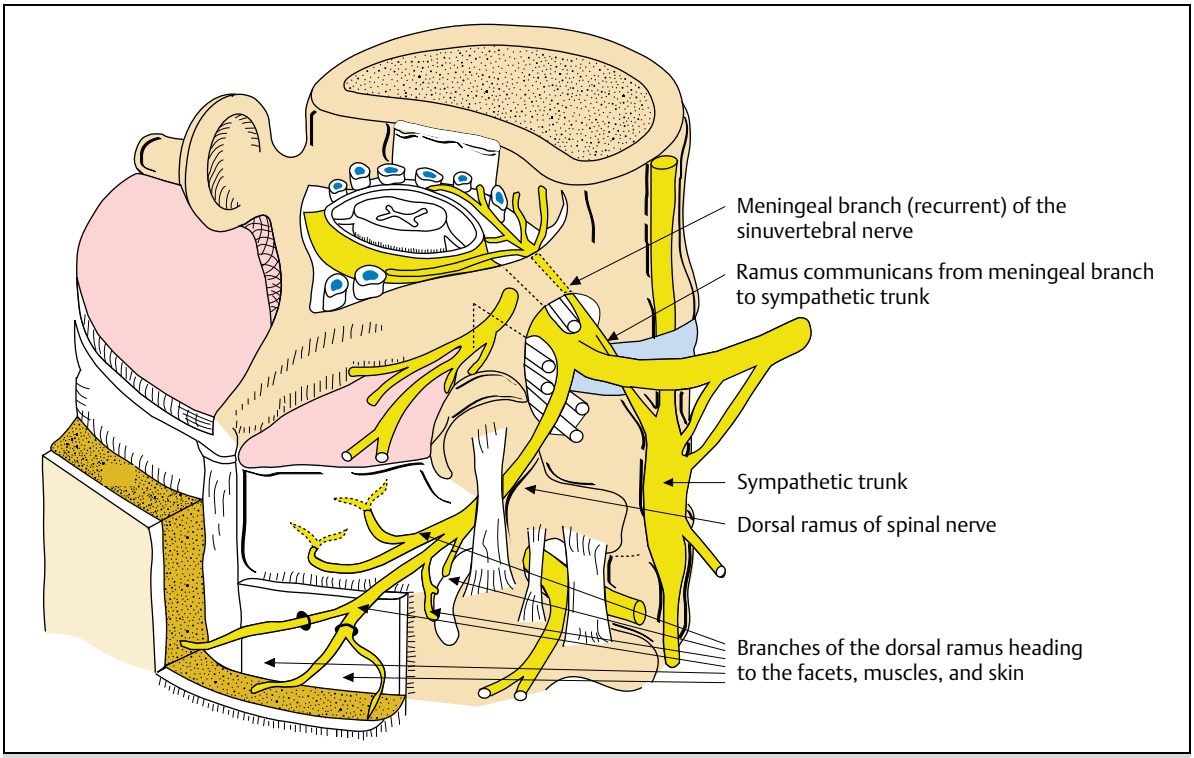


Fig. 9.3 The spinal nerve with its branches (from: Bogduk 1997).

ticular region of the inferior lumbar vertebral motor segments. Nociceptors in the joint capsule, the posterior longitudinal ligament, and the periosteum of the vertebral body are located in close proximity to the afferent fibers of various nerves. Disorders of form and function in the musculoskeletal system cause reactive changes in the joint capsule and outer edges of the vertebral body as a result of intervertebral disk degeneration. At the same time, this causes irritation of the nociceptors found there, leading to neuralgia when afferent fibers are irritated over a longer period of time. The autonomic reaction is directly and indirectly involved as a result of the irritation of the ramus communicans from the meningeal branch to the sympathetic trunk and via the spinal nociceptive reflex arc (see Chapter 1, " Moving from Acute to Chronic Pain: Nociceptor Sensitization").

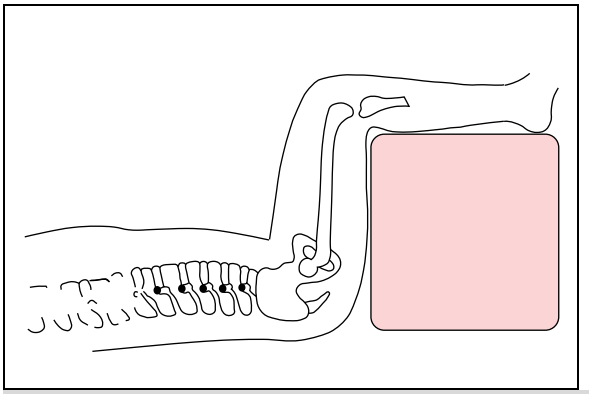
The foraminal articular region of the lower lumbar vertebral motor segments is particularly important in the development and treatment of chronic back pain and sciatica. Obviously the nociceptive neuralgic structures, located so close to each other, are suited for local treatment, e. g., infiltration.

Basic Therapy for Lumbar Pain

General therapeutic measures, such as bed rest, heat treatment, massage, electrotherapy, and analgesics, act in some way on the vicious cycle of pain–tense muscles–pain, resulting in freedom from pain in mild cases. The lumbar intervertebral disks experience the least amount of loading when the patient is positioned horizontally with the lumbar lordosis flattened out and the hip and knee joints flexed. For this reason, the use of the so-called **Fowler position** is recommended as the first treatment measure (**Fig. 9.4 a, b**).

Heat, in the form of mud packs, heat pads, or infrared treatment, acts to relieve pain and muscle tension. **Analgesics and anti-inflammatories** are additionally administered when pain is very severe (see Chapter 4, "Multimodal Medication Concomitant Therapy").

Massage and electrotherapy are first administered in acute lumbar syndromes after the acute symptoms have been largely relieved by positioning, heat treatment, and analgesics. Electrotherapy is administered using high frequency, low frequency, and interferential currents (see Chapter 4, "Electrotherapy").



a

b

Fig. 9.4 a, b The Fowler position: Hip and knee joints are placed at right angles, in a step-like manner. The sciatic nerve is maximally relaxed in this position. The lumbar lordosis is flattened, which in turn widens the intervertebral foramina and the lumbar vertebral canal. The zygapophyseal joint capsules are relieved of loading.

Caution: When mobilization is difficult always think of thrombosis prophylaxis, using low-dose heparin and antithrombotic stockings.

Ultrasound relaxes ligamentous and insertion tendopathies, especially in the region of the interspinous ligaments and the short back muscles. **Diadynamic currents** have a deep analgesic effect at the nerve roots and their branches. Deeper-lying spinal structures can also be reached by the use of **short wave** radiation.

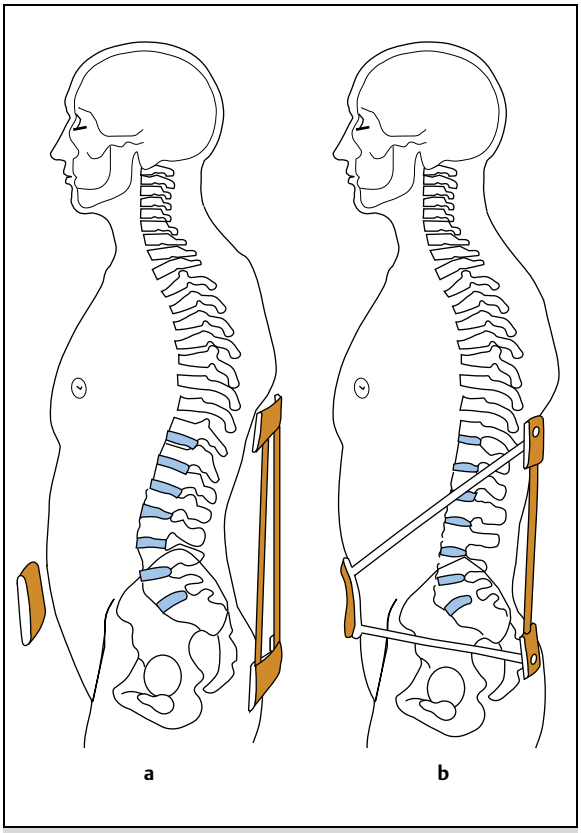
Manual therapy is indicated in the treatment of lumbar spinal pain when acute local zygapophysial joint or sacroiliac joint symptoms are present, or when another functional disorder is the focus of attention. This is precisely ascertained using manual assessment techniques. Manual therapy should be used with caution when intradiscal mass displacement, protrusions, and prolapses are present as manipulation can sometimes increase the degree of protrusion.

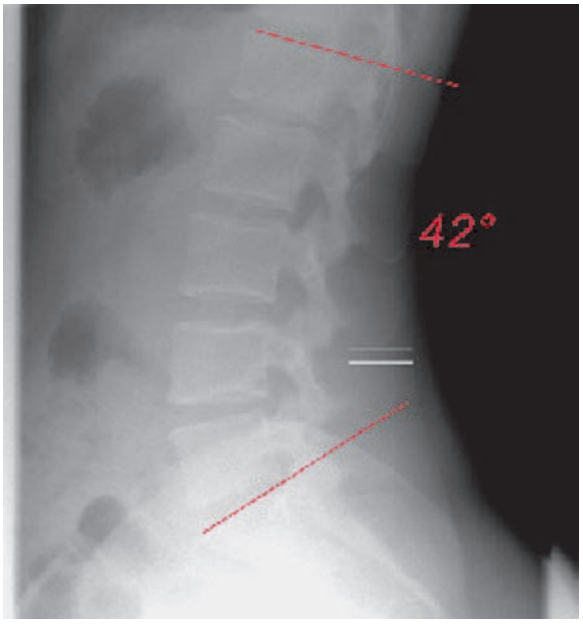
If the anulus fibrosus is intact, the use of **traction** enables displaced intervertebral disk tissue to return to the center of the disk. Several options are available for traction in the lumbar spine region, including self-traction, permanent traction devices positioned on the iliac crest, or traction belts (Kraemer 2009). The physician fits these orthopedic aids individually according to how treatment is progressing, on the basis of clinical neurological signs which must be reassessed at regular intervals.

Traction on an inclined bed or traction table, or using Perl's apparatus, has now been replaced by the more manageable **flexion cube**, which repositions lumbar intervertebral disk protrusions in the same way, combined with heat treatment.

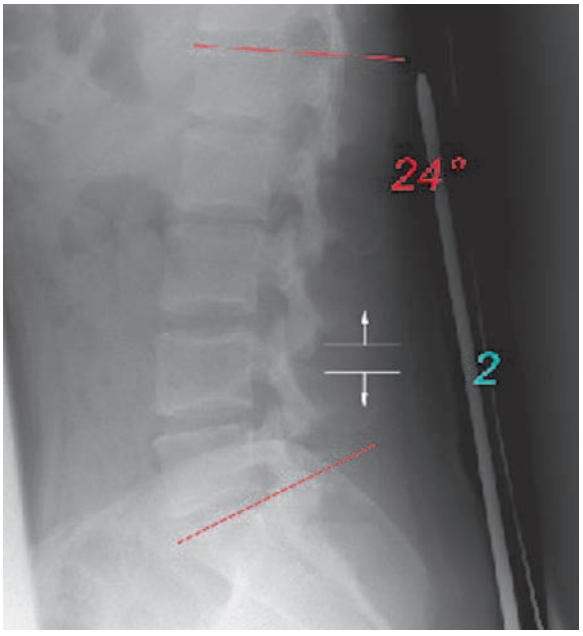
The supportive and corrective function of **flexion orthoses** is useful in the treatment of acute and chronic pain in the lumbar spine that is caused by the displacement of intradiscal mass and a hyperlordotic position of the zygapophyseal joints. Their use is also indicated in postsurgical segmental instability, e. g., following discotomy, percutaneous nucleotomy, or chemonucleolysis. Degenerative instability with hight reduction in the posterior section of the vertebral motor segment is also an indication for the use of orthoses, which should reduce loading on the vertebral motor segment by increasing intra-abdominal pressure and flattening out the lumbar lordosis. This flattening leads to widening of the intervertebral foramina and the spinal canal (**Fig. 9.5a–d**).

During **physiotherapy**, pain is relieved by using traction in a neutral position. This is usually conducted in a relaxed position with the legs raised in the Fowler position (**Fig. 9.6**). When acute pain is present, this is in fact the only possible starting position. The patient is placed in this initial position and attempts to move normally and to stabilize the affected spinal region at the same time. As the symptoms start to diminish, other starting positions can be used until the lordosis is normal again.





c



d

Fig. 9.5a–d Flexion orthosis used in the treatment of acute and chronic pain in the lumbar spine caused by a narrowing of the posterior intervertebral disk height and compression of the zygapophyseal joint. The effect is obtained by using an orthosis with a suprapubic abdominal pad (**a** and **b**) which pushes the abdomen in. The straight back section flattens the lumbar lordosis (**c** and **d**). The increase in intra-abdominal pressure lowers the intradiscal pressure by approximately 30 %.

Movement Program (MIPFR) for the Lumbar Spine

The first rule of back school is, "Keep moving." With this in mind, all types of movement that do not provoke back pain are appropriate, based on the movement in pain-free range (MIPFR; see Chapter 3.5) principle. Central to the movement program for chronic back pain are back-friendly sports which use linear movements, e. g., swimming, jogging, and cycling. Other patterns of movement are also appropriate as long as the patient does not adapt an excessively lordotic posture or thoracic kyphosis combined with rotary movements. The sports or types of exercise recommended in the individual treatment program should be ones the patient mentioned as favorites in their initial assessment, so that exercises are conducted daily and regularly.

Fig. 9.6 Proprioceptive neuromuscular facilitation (PNF) treatment in a pain-relieving position (treatment cube).

Special Therapy for Lumbar Pain

Local Pain Syndromes in the Lumbar Spine (Local Lumbar Syndrome, Nonspecific Back Pain)

By definition, the pain caused by local lumbar syndromes is restricted to the lumbosacral region, i. e., there is no radiation into the lower limbs. The back pain is felt at its point of origin, so we are dealing with a **nociceptively governed clinical picture**.

The pain arises in the nociceptors of the zygapophyseal joint capsules, the posterior longitudinal ligament, and the interspinous ligaments. The nociceptors found in the muscle attachments and the long and short back muscles themselves are secondarily affected. The sensitive fibers of the meningeal branch and the dorsal ramus of the spinal nerve are predominantly affected. The permanent reflex tension in the back extensor muscles is perceived as unpleasant and painful. These patients suffer from position-dependent back pain, muscle tension, and restricted mobility of the lumbar spine. The different symptoms found in the local lumbar syndrome are often summarized in the term "**nonspecific back pain**," especially when the examiner is unable to assess precisely where the pain is originating from.

In order to target the local treatment of back pain, it is important to know where the pain originates, i. e., the zygapophyseal joint, back muscles, or sacroiliac joints. Important information is gained from the medical history and the results of the manual therapy assessment. Many investigators have indicated the importance of the zygapophyseal joints in the development of back pain (Ghormley 1933, Badley 1941, Mooney 1976, McCall et al. 1979, Carrera 1980, Young 1983, Law et al. 1985, Moran et al. 1988).

The symptoms can begin suddenly, e. g., when the trunk is abruptly rotated, or may arise gradually without any special reason. During the subjective assessment patients often mention exposure to cold or remaining in the same posture for some time among the precipitating factors. During the examination, the patient can quite clearly localize where the pain is coming from, in the initial stages. Pain is usually felt more on one side in the region of the sacroiliac joints and the lumbar back extensors. This is in the area supplied by the posterior branch of the L5 and S1 nerve roots. Permanent irritation of the dorsal ramus results in the development of a **neuralgia-governed component**, where pain spreads into the proximal sections of the lower limb and cannot be allocated to a specific segment, e. g., in the buttock region. Patients present with the typical tender points at the spinous processes and along the posterior section of the sacroiliac joints. In addition, the back extensors are distinctly under a certain amount of tension and the mobility of the lumbar spine is restricted. When acute pain syndromes are present, a motor reaction causes the patient to immediately adopt a posture of slight trunk flexion and, in some cases, lateral flexion (**Fig. 9.7**). This posture should not be altered, as it represents a **protective reflex** which aims to prevent further irritation of the nociceptors in the zygapophyseal joint capsule and the posterior longitudinal ligament.

Treatment of Acute Low Back Pain

The main aim of treatment in acute local lumbar syndromes is the immediate removal of pain, interrupting the chronification process right from the start. Peripherally acting **analgesics** (see Chapter 4, "Multimodal Medication Concomitant Therapy") block nociception and the distribution of pain signals at their point of origin. The simultaneous administration of **local infiltrations** at the source of pain is recommended. The source of pain is ascertained during the manual therapy assessment and by trial injections. The interspinous ligament insertions, usually between L4/L5 and L5/S1, as well as the zygapophyseal joint capsules, are suspected when acute arthroligamentous back pain is present. When pain is felt more on one side, the back extensors tense up asymmetrically and one sacroiliac joint is also often included in the primary painful event. Immediate mobilization (**manual therapy**), with the support of local infiltration with local anesthetics and concurrent electrotherapy, prevents a permanent blockage in the affected zygapophyseal and sacroiliac joints.

In this phase patients immediately accept the rules of back school, as incorrect posture and behavioral patterns immediately cause severe pain. Patients should be familiarized with these rules from the very beginning to prevent recurrence of pain and the evolution of chronic pain. As long as they do not attempt heavy physical labor, even patients with acute low back pain can pursue their accustomed way of life with only slight restrictions if they follow the postural and behavioral guidelines learned from back school. They can continue to walk, sit, stand, and carry out tasks involving light to moderate physical labor.

Controlled studies into acute low back pain (Coomes 1961, Gilbert et al. 1985, Postacchini et al. 1988, Deyo et al. 1991, Szpalski and Hayez 1992, Malmivaara et al. 1995, and Wilkinson 1995) have demonstrated that bed rest and immobilization impede the healing process rather than helping it. Therapeutic approaches that use centrally depressing medication are therefore obviously inappropriate. The emphasis should be on interventions that affect the nociceptive region locally. Treating physicians should choose the method that they are most familiar and comfortable with from the range of therapies that conform to this concept (**Table 9.1**)

Fig. 9.7 The adaptive posture seen in an acute lumbar syndrome (lumbago) with the trunk slightly bent forward and laterally flexed.

Table 9.1 Pain Therapy for Acute Low Back Pain

Analgesics
Pain-relieving position
Cryotherapy (heat therapy)
Local infiltrations (trigger points, facets, SIJ)
Manual therapy (traction)

Treatment of Chronic Low Back Pain

Pain that endures for weeks or months is at risk of becoming chronic. Patients find it more difficult to retain their accustomed way of life, and psychological disturbances are inevitable. The pain changes in character and is permanently present, sometimes even during the night.

Initially, the pain is selectively located at a zygapophyseal joint or in the area surrounding a sacroiliac joint. This develops into a diffuse pain in the lower back that spreads over the entire lumbosacral region. It can spread into one or both limbs as a pseudoradicular form of pain. The permanent tension in the back extensors and the proximal limb muscles causes insertion tendopathies at the pelvis, the spinous processes, and eventually also in the upper sections of the trunk.

Treatment consists primarily of heat treatment, exercises, and local infiltration at the initial and secondary sources of pain. Analgesics and sedatives should be administered with caution in cases of chronic low back pain, as side effects are more likely when they are used long term.

Treatment for a chronic lumbar syndrome aims primarily to break the vicious cycle of pain–cramped muscles–

Table 9.2 Pain Therapy for the Chronic Lumbar Syndrome

Heat
Local injections
Movement therapy
Back school
Psychotherapy
Progressive muscle relaxation

Fig. 9.8 The typical adaptive posture for acute sciatica on the right-hand side.

adaptive posture–pain at the muscular level. Massage, electrotherapy, infiltration of muscles using local anesthetics, and general measures used to relax muscles are ideal for this purpose. Progressive muscle relaxation according to Jacobson (1938) is the primary intervention used to achieve this. The increased muscle tension originally served a useful purpose, but as the chronically recurring lumbar syndrome progresses it becomes an independent part of the vicious cycle. This results in pain caused by constant muscle tension and insertion tendopathies.

When the pathological muscle tension has eased as a result of heat treatment, electrotherapy, or distraction, the use of exercise is appropriate provided it is performed carefully and in the pain-free range of movement. Possibilities include stationary cycling and swimming backstroke in a stress-relieving posture. Back school postural and behavioral training dictates that all movements and postures that can result in new nociceptive input must be completely avoided. In the chronic lumbar syndrome, this mainly means avoiding extension and rotary movements of the lumbar spine, excessive kyphotic postures, or maintenance of the same posture for a long time. The exposure of muscles to cold, or psychological stress, can also assist the chronification process (**Table 9.2**).

Pain Therapy for the Lumbar Nerve Root Syndrome

Dermatomal spread of pain into the leg indicates the irritation of a spinal nerve, with the involvement in particular of the ventral ramus. This is a **neuralgia-governed clinical picture**. Generally speaking, the spinal nerves belonging to the two most inferior lumbar vertebral motor segments are affected and patients present with symptoms of sciatica.

Lumbar nerve root syndromes are caused by intervertebral disk protrusions or prolapses and/or bony impingement in the lateral recess and in the intervertebral foramen. The clinical symptoms associated with discogenic sciatica arise suddenly in most cases and quickly adopt neuralgic characteristics. The afferent fibers in the spinal nerve are rapidly converted to nociceptors. Pain that is position-dependent, accompanied by a feeling of numbness or ants crawling along the dermatome, is characteristic of this disorder (**Fig. 9.8**). The neuralgia can be accentuated by external factors such as axial loading, sudden changes in posture, and bending the trunk.

In the lumbar nerve root syndrome **pain chronification** is predetermined because of the neuralgia. The lumbar nerve root syndrome is a primary chronic disorder: The conversion of a primary conducting nerve to a nerve with a nociceptor function already signifies chronicity. The longer the condition remains, the less chance there is that simple forms of intervention, such as manipulation, traction, or a nerve root block, can rapidly improve the pain. The constant irritation of the spinal nerve root leads to secondary symptoms with motor and autonomic reactions. In the course of time, central changes occur in the perception and processing of pain. In some cases, characteristics such as different positions affecting pain levels and the day–night pattern can no longer be observed: Pain is felt permanently, and associated psychological symptoms appear. The constant maladaptive postures cause muscle tension and insertion tendopathies not only in the lumbar region, but also in the upper sections of the trunk.

Pain therapy for the lumbar nerve root syndrome targets not only the peripheral nociception, but also the transmission and processing of pain signals; local injections using lumbar spinal nerve analgesia and epidural injections are the main focus. In both acute and chronic

lumbar nerve root syndromes it is advisable to tackle the point of nerve root compression directly (see Chapter 11).

Pain Therapy for Lumbar Spinal Canal Stenosis

The characteristic pain arising from a lumbar spinal canal stenosis varies according to the amount of weight-bearing when walking and standing, and is reduced by all postures involving trunk flexion (flattening out the lordosis). This differs from the ischemic pain caused by circulatory disorders. Leg pain is the main symptom. It presents as bilateral pain that is found especially along the anterior side of the thigh in the case of **central spinal canal stenosis** and as an ipsilateral dermatomal spread in the case of **lateral spinal canal stenosis**. The leg pain often develops after walking for only a few steps, and is therefore also described as **intermittent spinal claudication**.

Possible causes for the constriction in the lumbar vertebral canal include bony involvement (vertebral arch, vertebral body) as well as the involvement of soft tissue structures (intervertebral disk, connective tissue). Spinal canal stenosis can occur in one segment or in several segments, depending on the cause. In most cases, the epidural space is characteristically narrowed by the ligamentum flavum protruding from the posterior side and the degenerative changes found in the intervertebral disk from the anterior side (**Fig. 9.9**). Both of these protrusions develop as part of the degenerative narrowing of the intervertebral section. Hyperlordosis amplifies these phenomena. These deformations are initially symptom-free and are described as **compensated spinal canal stenosis**. An increase in the lumbar lordosis due to age-related abdominal muscle weakness, small amounts of intradiscal mass displacement with the amplification of one or more intervertebral disk protrusions, and postural and behavioral changes with an associated increase in the lumbar lordosis additionally narrow the space available for the neural elements in the lumbar vertebral canal. This continues until there is no more space available and leads to the development of a compression-related pain.

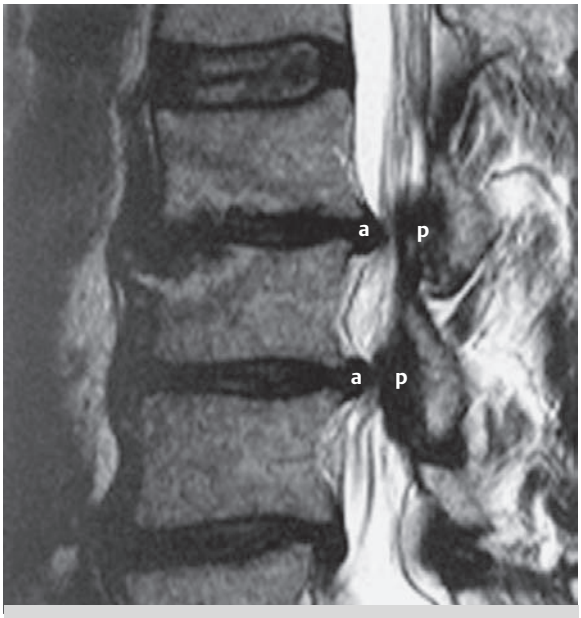


Fig. 9.9 Sagittal MRI section of the lumbar spine; spinal canal stenosis at two levels with protrusion of the intervertebral disks from anterior (a) and the ligamentum flavum from posterior (p).

Decompensated Spinal Canal Stenosis

Compression-related nerve root edema and congested epidural veins lead to the rapid development of spinal canal syndrome, which is accompanied by severe pain in both legs. This can occur in one segment or several. As it is mainly spinal nerves that are being compromised, the pain is dominated by neuralgia. According to Porter (1985), the neurogenic intermittent claudication associated with spinal canal stenosis never exceeds a certain level and does not result in paraplegia.

Because of the neuralgic character of the pain, centrally acting analgesics are administered as part of the symptomatic pain therapy for lumbar spinal canal stenosis with the aim of maintaining the mobility of the (mainly older) patients. The disorder of venous drainage in the spinal canal should be treated with agents that stimulate blood flow. The spinal nerve roots that are compromised in the vertebral canal are best reached by the use of epidural injections. In the case of a **central spinal canal stenosis** involving several segments, the use of a **posterior epidural** injection is appropriate as several segments can be reached at the same time. When patients present with a **lateral spinal canal stenosis** with compression of a single spinal nerve in the lateral recess or in the intervertebral foramen, an **epidural perineural** injection is indicated. Spinal nerve analgesia and facet infiltration are administered to complement the treatment, addressing the hyperlordosis and the tense lumbar muscles.

Causal pain therapy aims to flatten out the lumbar lordosis and thus significantly widen the lumbar vertebral canal. This additionally flattens intervertebral disk protrusions on the anterior side of the spinal canal and the protruding ligamentum flavum on the posterior side. The lumbar lordosis immediately flattens out when the patient is placed in the Fowler position and wears a flexion orthosis while standing and walking. Physiotherapy focuses mainly on strengthening the abdominal muscles. Twice daily stationary cycling (mornings and afternoons for 30 min) complements the physiotherapy program when conducted as MIPFR (**Table 9.3**).

Surgical widening of the lumbar spinal canal is an option when the disorder is resistant to treatment. It was common in the past to widen the spinal canal using a

Table 9.3 Pain Therapy for Spinal Canal Stenosis

Symptomatic	*Causal*
Psychological pain therapy Central analgesia	Fowler position
Stimulation of venous blood flow	Physiotherapy from the pain-relieving position
Epidural injection	Stationary cycling (MIPFR)
Spinal nerve analgesia	Flexion orthosis
Facet infiltrations	Decompression surgery

Fig. 9.10 MRI scan of a patient suffering from a postdiscotomy syndrome following an open lumbar intervertebral disk operation at the L4/5 segment. A surgical scar can be seen on the right-hand side. It extends continually from the skin, via the subcutaneous tissue and the back extensors, into the inner space of the lumbar vertebral canal. The scar tissue then extends further, passing between the dura and the vertebral canal, affecting the traversing and exiting nerve roots, and extends as far as the intervertebral disk. Movement of the (degenerative or surgically) unstable intervertebral segment is transferred anteriorly directly to the scar and the entrapped neuropathically modified nerve root. Likewise, movement in the back muscles is transferred posteriorly to the nerve root by the pull on the scar.

laminectomy over several levels. Nowadays, surgical intervention is limited to microdecompression at the affected segment. This is based on the knowledge that spinal canal stenosis is caused only by intervertebral disk involvement or the narrowing of a recessed area.

Pain Therapy for Problem Patients Who Have Undergone Spinal Surgery

These patients still have back and leg pain even after one or several intervertebral disk operations or surgical fusions. Chronic pain arises both from the peripheral nociception in the vertebral motor segment and in the neurologically modified nerve fibers. There is therefore a mixture of nociceptive and neuralgia-governed pain symptoms. The particular characteristic of pain chronification in postsurgical patients is that the actual operation on the spine (either open intervertebral disk surgery or spinal fusion surgery) involved nociceptors that were already sensitized and nerve fibers that were already modified into nociceptors. Direct intraoperative trauma to the nociceptors and the neurologically modified nerves in the area of the wound has led to further permanent damage. Postsurgical scar tissue is formed, with the involvement of neural structures that have previously been neuropathically damaged (**Fig. 9.10**). These neuropathically modified nerves and sensitized nociceptors, which are repeatedly irritated by tension in the scar tissue, form the pathological and anatomical substrate for chronic pain in patients who have already undergone spinal surgery.

Character and Severity of Pain

The pain in these cases is characterized by a bilateral, mixed pseudoradicular/radicular set of symptoms. Frequently, several nerve roots are involved. Neurological deficits may also be the result of previous operations and should not always be attributed to the current clinical picture. Severe neurological disorders are quite rare: Although the nerve root is strangulated by the scar tissue, it is not completely severed. Pseudoradicular components and nociceptively amplified pain are the result of segmental instability with irritation of the zygapophyseal joint capsule and the nociception in the posterior longitudinal ligament. These patients have only a small pain-free range of movement because spinal nerve roots and their branches, now partly fixated by scar tissue, have been partially severed during surgery leaving free nerve endings.

NOTE

The strands of connective tissue along the dura and nerve root can be compared to wind chimes that are stirred into action by any kind of careless movement.

Table 9.4 Symptoms of Postdiscotomy Syndrome
Bilateral, mixed pseudoradicular/radicular symptoms
Double-sided positive Lasègue test
Impossible to flex trunk or sit with legs extended
Epidural scarring seen on MRI and CT

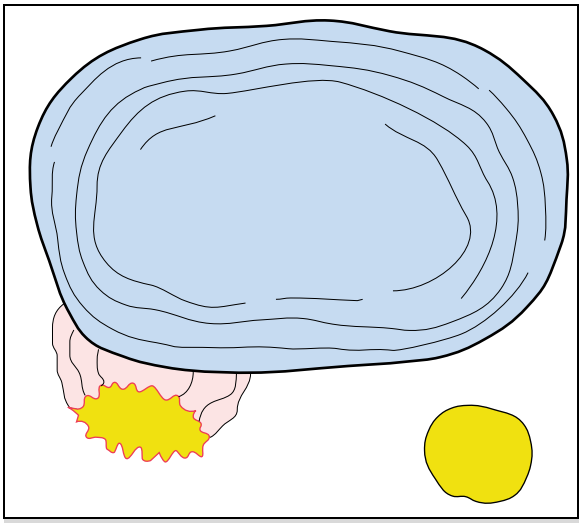


Fig. 9.11 The slightest change in the volume and consistency of the intervertebral disk causes a reaction in the disk and the nerve root because these two structures are connected by scar tissue.

Nociceptors and afferent fibers are in a permanently irritated state. Inflammatory and edematous processes cause swelling in the spinal nerves and further narrow what space is left in the vertebral canal. A vicious cycle begins. The mobility of the sciatic nerve root during trunk flexion is most affected. This is noticeable when the leg is extended and raised, or when the patient sits with straightened legs. In severe postsurgical pain syndromes, the Lasègue test is bilaterally positive after only 10–20° of movement. The mobility of dura and nerve roots in the vertebral canal is often so limited that even flexing the neck aggravates the typical range of symptoms (**Table 9.4**, **Fig. 9.11**).

Patients with a pronounced postdiscotomy syndrome following one or several intervertebral disk operations are considerably impaired: They are unable to sit, stand, or lie down normally (**Fig. 9.12**). As they have no serious neurological deficits, these patients are often labeled as "pension neurotics" or categorized as suffering from psychological overlay.

The **severity** of a postdiscotomy syndrome is primarily determined by the subjective level of impairment. Objective criteria, such as neurological deficits, scarring, and instability, are not always an accurate measure of severity. Several consultations with the patient may be necessary to define their amount of suffering, with different people observing. The straight-leg-raise test in lying and sitting is relatively reliable, as is observing the patient when they sit with their legs extended, take off and put on their shoes and socks, and flex the trunk during other activities (**Table 9.5**).

Fig. 9.12 A patient with a pronounced postdiscotomy syndrome following several intervertebral disk operations, who has considerable functional limitations in daily life and can mobilize only with the use of walking aids.

Therapy Approaches

The **pain therapy approach** for problem patients who have previously undergone back surgery is multifaceted. It corresponds to the patient's mixture of nociceptive and neuralgic symptoms and the associated significant changes in pain transmission and perception. Either peripherally or centrally acting analgesics are administered, depending on whether the nociceptive or neuralgic component is more prominent. As the centralization process is usually quite advanced, most patients report that only centrally acting analgesics bring relief (Theodoridis et al. 2004).

Local injection therapy is used to directly influence the nociception and neuralgia in the previously operated vertebral motor segment. The use of epidural injections and

Table 9.5 Level of Severity of Postdiscotomy Syndrome (According to Krämer 2009)

Level	*Pain*	*Lasègue test*	*Medication*	*Functional capabilities*	*Expert opinion*	*DG* in %*
I	No resting pain, mild pain on loading	Negative	Occasionally	Restrictions for heavy physical labor and competitive sport	able to work, but no heavy physical labor	under 20
II	Mild resting pain, severe pain on loading	Positive	Regularly weak, occasionally strong	No work that places load on the spine, no sport	Often unable to work, occupational disability for work that loads the spine	30–80
III	Severe permanent pain	< 30°	constantly severe	Walking aids, requires assistance from others	General disability	100

*

Table 9.6 Pain Therapy for Problem Patients Who Have Previously Undergone Spinal Surgery

Symptomatic	*Causal*
Analgesics (central and peripheral)	Stabilization of the trunk muscles, physiotherapy
Psychological pain therapy	Psychological pain therapy
Local injections	Back school
Alternative medicine	Orthosis (temporary)
Movement therapy	Spondylodesis

spinal nerve analgesia at the affected segments is well established. When administering interlaminar epidural injections using the loss-of-resistance technique, injections must be placed one or two segments higher or access obtained via the sacral canal (as an epidural sacral injection). This is necessary because of the adhesions found in the epidural space at the surgical site. The epidural perineural injection technique is the best way of directly influencing the entrapped, edematously swollen nerve root. Intradiscal injections may be considered if the postsurgical MRI or CT still shows a broad-based dent caused by an intervertebral disk protrusion.

Chronic pain conditions originate mainly in the dorsal ramus and are treated with facet infiltrations and scar infiltrations.

To treat instability, the second pathogenetic component in the postdiscotomy syndrome, it is worth trialing the use of a trunk orthosis. Flexion orthoses that relieve loading on the posterior section of the vertebral motor segment seem to be appropriate. Concurrent physiotherapy with isometric stabilizing exercises starting in a pain-relieving position is essential. The trunk muscles are trained using intensive exercises. The ultimate aim is for these muscles to take over the function of the orthosis and contribute to the stabilization of the previously operated vertebral motor segments.

Multifaceted pain therapy is justified in the treatment of the postdiscotomy syndrome because of the syndrome's multifaceted etiology and pathogenesis and its equally multifaceted set of symptoms. All types of intervention that do not cause additional damage to the patient are important. The body's own pain-inhibiting mechanisms should be activated as much as possible. This includes all of the psychological measures available to cope with and reduce the pain, as well as the use of an individually tailored movement program (MIPFR; **Table 9.6**).

Pain Following Lumbar Spondylodesis

A posterior, anterior, or posterior/anterior spondylodesis at the affected vertebral motor segment is a surgical treatment option when all conservative measures that aim to stabilize the surgically unstable segment have failed. This operation is also performed when post-traumatic instabilities and deformations (e.g., spondylolisthesis) are present. The main indication worldwide is nevertheless the problem patient who has previously undergone one or more intervertebral disk operations. By eliminating the instability component, it is usually possible to relieve a large proportion of the pain in postdiscotomy syndromes. However, new pain symptoms often develop, originating in the neighboring segments or the sacroiliac joints. This pain complex is called the **postfusion syndrome** (Krämer 2009). The pain arising from the segment instability near the fusion site, the sacroiliac joints, and the new scar tissue is then combined with the residual pain coming from the postdiscotomy syndrome.

The **pain therapy approach** for this particularly problematic group of postsurgical patients is largely the same as for the postdiscotomy syndrome. The predominant set of local symptoms has to be identified using clinical neurological examinations and, in particular, trial local infiltra-

tions. The psychological care of these patients is particularly important. After undergoing repeated and sometimes very complicated operations, they are very skeptical about any type of medical intervention and tend to prefer long-term medication with centrally acting analgesics.

Exercise Program

The exercise program aims to make use of the limited pain-free range available. Back surgery patients with severe pain can once again exercise without running the risk of their pain increasing during or following activity—mainly swimming, cycling, and some suitable exercises. The sports instructor should focus on expanding the patient's possible movement spectrum by introducing him or her to other patterns of movement, such as jogging under minimal loading, aqua-jogging, Thera-Band exercises in a lying position, etc. For many patients, it is quite an experience to be able to move more without experiencing pain.

Lumbar Injection Therapy

Lumbar Spinal Nerve Analgesia

Principle

Posterolateral injection of a local anesthetic (mixed with steroids, when necessary) into the foraminal articular region of the vertebral motor segment.

Topographical and anatomical palpation points determine the injection angle and needle path. The lumbar spinal nerve analgesia differs from the Reischauer (1953) and Macnab and Dali (1971) techniques in that the needle is placed at an angle rather than in a sagittal direction. The injection site is 8–10 cm lateral to the midline and the needle is inserted at an angle of approximately 60°. Using this method, bony contact is always established with the posterolateral section of the lumbar vertebra.

Indication

All acute and chronic local and radicular lumbar syndromes are indications for lumbar spinal nerve alalgesia (LSPA). However, other forms of lumbar vertebral motor segment irritation caused by osteoporotic fracture, spondylolyses, tumor-related pain, spinal canal stenoses, and inflammatory pathological changes, particularly in the area of the vertebral joint capsule, also respond well to this method of treatment (**Table 9.7**).

Technique

The needle is 10–15 cm long (usually 12 cm), depending on the soft tissue depth. The intervertebral foramina of the inferior lumbar spine are best reached by inserting the needle 8 cm lateral to the midline at the same level as the iliac crests. The needle is placed at a 60° angle in the horizontal plane and at different angles in the vertical plane depending on which nerve root is affected:

- To infiltrate the **L3** nerve root, the needle is inserted at an angle of **0°**.
- To infiltrate the **L4** nerve root, the needle is inserted at an angle of **30°**.

The needle is inserted superior to the L5 transverse process for 1–2 cm until bony contact is made. The tip of the needle is then positioned on the lateral facet, immediately next to the intervertebral foramen, or along the side wall of the vertebral body. It is here that the ventral ramus, the efferent branches of the dorsal ramus, the meningeal branch, and the ramus communicans run down to the sympathetic trunk.

To infiltrate the **L5** nerve root when it exits the L5/S1 intervertebral foramen, the needle tip is further lowered underneath the L5 transverse process, corresponding to an angle of approximately **50–60°** in the vertical plane. The needle is inserted until bony contact is made with the lateral vertebral body or the lateral facet.

CT-monitored spinal nerve analgesia (**Fig. 9.13a–c**) has demonstrated that the injected solution disperses through the intervertebral foramen, additionally reaching the traversing S1 nerve root at the exact point where the L5/S1 intervertebral disk applies pressure onto the nerve.

It is possible for a root sheath to be punctured in the intervertebral foramen. For this reason, constant attempts to aspirate are made while the needle is being inserted, particularly during the final phase of the procedure. When contact is made with the nerve root, the patient indicates the presence of a sudden sharp pain radiating into the leg. This pain phenomenon can be largely prevented by proceeding slowly with continual pre-injection and aspiration. It is therefore recommended that a total of 10 mL of a

Table 9.7 Indications for Lumbar Spinal Nerve Analgesia

Local lumbar syndrome
Lumbar nerve root syndrome
Osteoporosis
Spondylolysis, spondylolisthesis
Tumor
Spinal canal stenosis
Rheumatic inflammatory changes to the vertebral column
Postdiscotomy syndrome

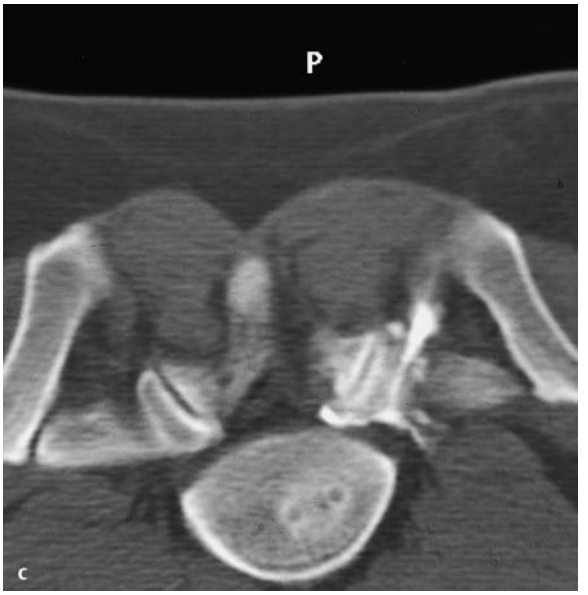


Fig. 9.13a–c Posterolateral L5/S1 perineural injection on the right. MRI scan of the cribriform fascia (arrow). This fascia distinguishes the perineural space from the deep autochthonous back muscles and the paravertebral fatty tissue (**a**). The contrast agent flows back into the needle when this fascia is not punctured (**b**). The contrast agent correctly spreads perineurally when the needle is inserted a further few millimeters and perforates the fascia (**c**).

dilute local anesthetic solution be used for the injection, as in the end usually only 4–5 mL are available for the actual injection at the target site. Once the final position of the needle has been correctly established, it is possible to add a longer-lasting local anesthetic (e. g., bupivacaine) and/or a glucocorticoid (e. g., 10 mg triamcinolone) depending on the presenting clinical situation (**Fig. 9.14**).

Effects of Lumbar Spinal Nerve Analgesia

Despite the posterolateral administration of local anesthetic, the nociceptors found in the posterior longitudinal ligament, the posterior anulus fibrosis, and the zygapophyseal joint capsule can be reached indirectly via the meningeal branch. As shown in our CT investigations using contrast agents, a portion of the injected solution disperses to the proximal parts of the spinal nerve, reaching both the spinal ganglion and the ramus communicans. Only a portion of the injected solution reaches the epidural space via the intervertebral foramen.

Following the administration of paravertebral lumbar spinal nerve analgesia, the patient feels a decrease in the back and leg pain, which is maintained for an average of 3.5 hours when a 0.5–1 % local anesthetic solution has been used. Approximately 50 % of the patients surveyed by us (Krämer 2009) also indicated a distinctive feeling of relax-

ation and a subjective sensation of warmth in the back and the affected leg. Temporary signs of paralysis or feelings of lameness in the leg are to be expected in 8 % of all cases. Patients must be made aware of this in advance and precautions must be taken accordingly.

NOTE

The aims of LSPA are not complete analgesia and paralysis of lumbar spinal nerves, as is the case when preparing for surgery, but rather the reduction of pain and the desensitization of irritated neural structures in the lumbar vertebral motor segment.

Lumbar Spinal Nerve Analgesia Injection Procedure (Figs. 9.14–9.48)

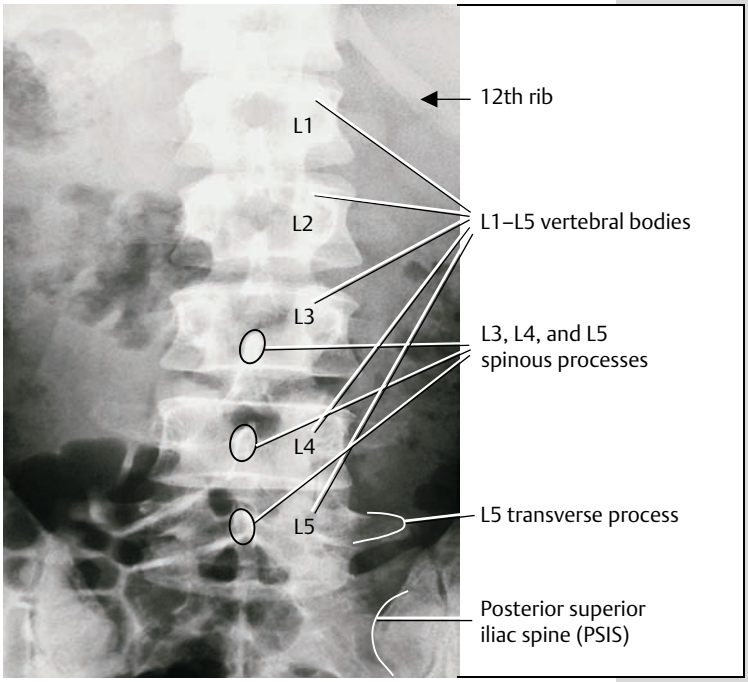


Fig. 9.14 The anteroposterior lumbar spine radiograph is displayed the right way around (right = right) in the field of vision of the practitioner, who assesses and palpates the sitting patient's inferior lumbar region from a posterior direction. Note changes in the anatomical orientation points when scolioses, junctional anomalies, and numeric variations in the number of lumbar vertebrae are present.

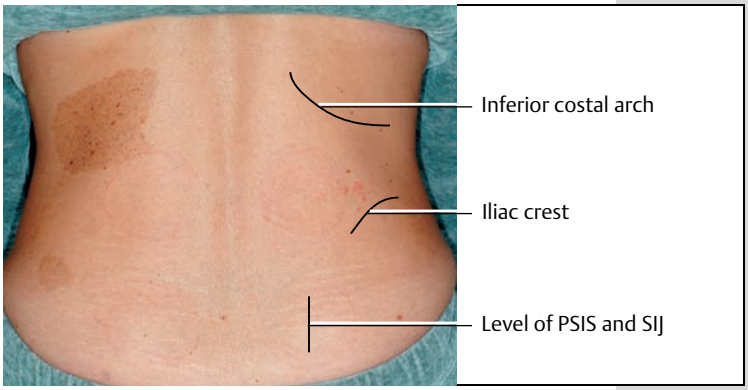


Fig. 9.15 The inferior lumbar region from the point of view of the seated practitioner. The patient's back is free of clothing so that the inferior costal arch, the waist, the posterior superior iliac spines (PSIS), and the sacroiliac region (SIJ) can be easily assessed and palpated. The skin must be intact and show no signs of infection.

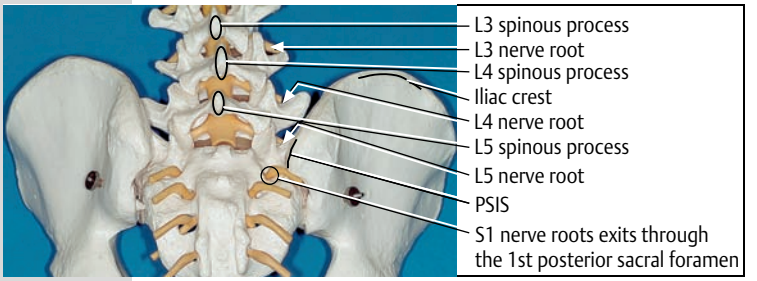


Fig. 9.16 The most important anatomical orientation points demonstrated on a skeleton. The iliac crest and spinous process are always palpable on patients sitting slightly flexed, even on obese patients. These palpatory anatomical orientation points are related to the facets and the exiting nerve roots.

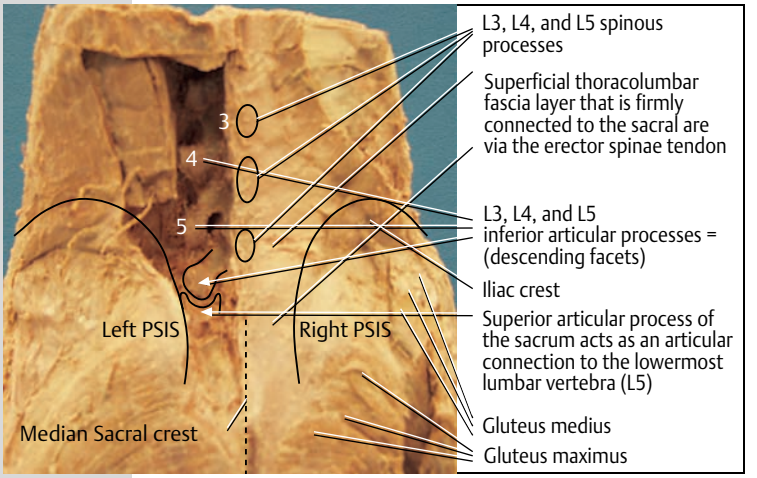


Fig. 9.17 The proportions and distance between the iliac crest and L3/4/5 spinous processes can be seen on this specimen. The LSPA needle has to pass through the relatively large posterior trunk muscles to reach the foraminal articular region of the lower lumbar vertebral motor segments.

Fig. 9.18 The examiner is positioned directly behind the patient, who is sitting on a higher examination couch. The physician's assistant stands in front of the patient. Oxygen saturation and pulse frequency are monitored using a pulse oximeter. The treating physician or the assistant constantly monitors the patient verbally throughout the procedure. The examiner must be comfortably able to bimanually palpate the iliac crests and the border of the sacroiliac joints. This arrangement is used for the LSPA. The thumbs slide medially from a lateral position, over the posterior iliac spine in the sulcus between the iliac spine and the medial sacral crest. The index and middle fingers palpate the iliac crest (from Theodoridis T, Ludwig J, and Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

Fig. 9.19 Bimanual palpation of the posterior superior iliac spines and the iliac crests demonstrated on a skeleton.

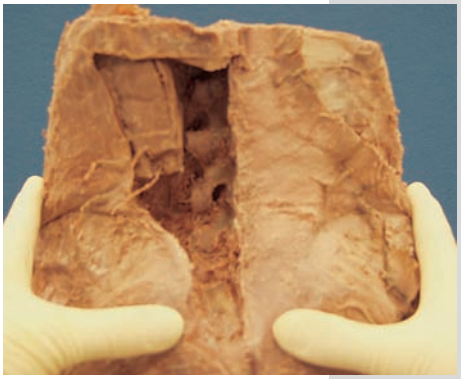


Fig. 9.20 Bimanual palpation of the posterior superior iliac spines and the iliac crests demonstrated on an anatomical specimen.

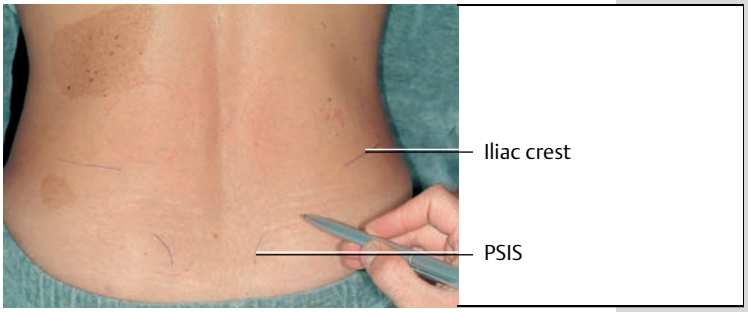


Fig. 9.21 Marking the palpation points along the iliac crest and the posterior superior iliac spine.

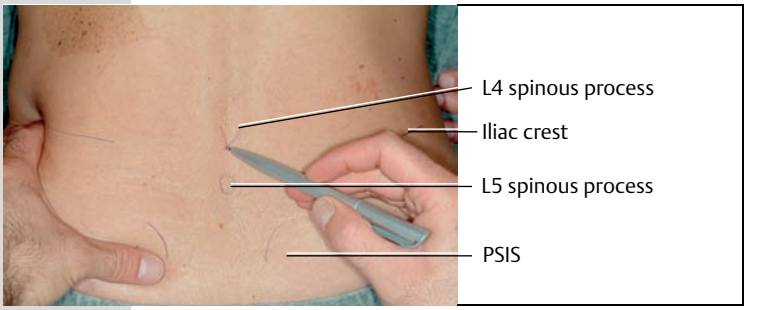


Fig. 9.22 Marking the tips of the L4 and L5 spinous processes in relation to the iliac crest. The connecting line between the iliac crests corresponds to the height of the L4 spinous process (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

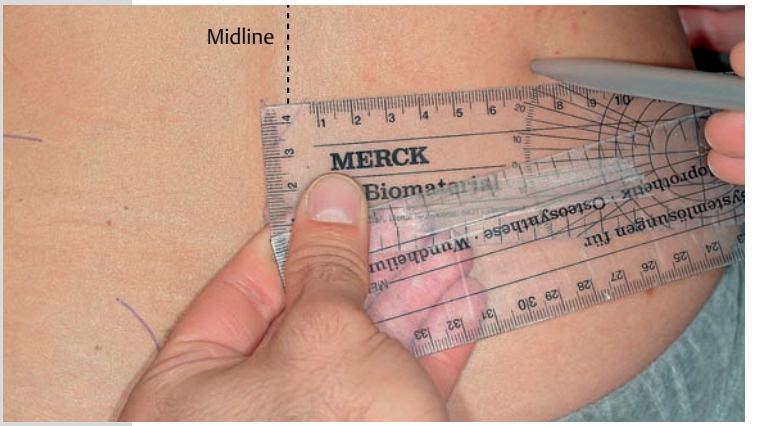


Fig. 9.23 The intervertebral foramen of the lower lumbar spine is best reached using an injection site ~8 cm lateral to the midline at the same level as the iliac crests. The location of the injection site has to be individually selected for each patient depending on the width of the trunk; 8 cm is an average value. A pen with retracted ink cartridge is used to mark the injection site: The marked location remains visible as a dent after disinfection.

Fig. 9.24 Towels are placed over the patient's clothing. The skin is disinfected by spraying with disinfectant several times. Before positioning the needle at its final angle in the horizontal plane (see **Fig. 9.27**), it is recommended to place the needle at the marked point vertical to the skin without inserting the cannula.

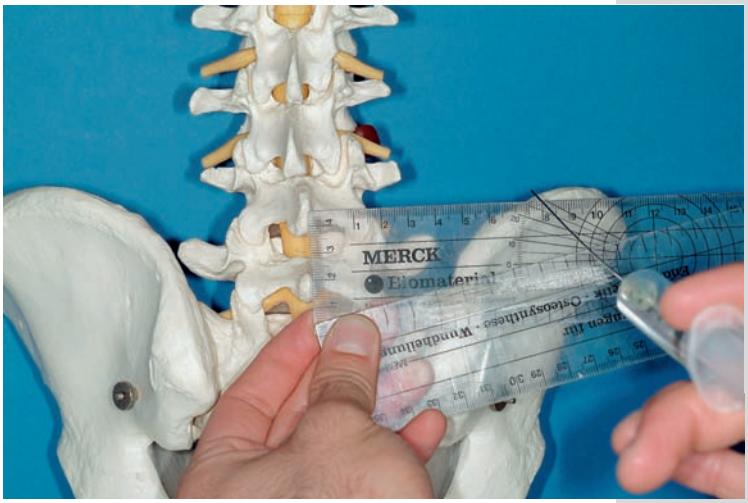


Fig. 9.25 Demonstration on a skeleton: Access to the foraminal articular region for the L3, L4, L5, and S1 nerve roots is found at an average of 8 cm from the midline, directly above the posterior section of the iliac crest.

Fig. 9.26 Demonstration on an anatomical specimen: The practitioner must ensure that the insertion site is still above the iliac crest, by palpating deeply. This is necessary so that the foraminal articular region is reached, also at L5/S1, when the needle is later positioned at an angle.

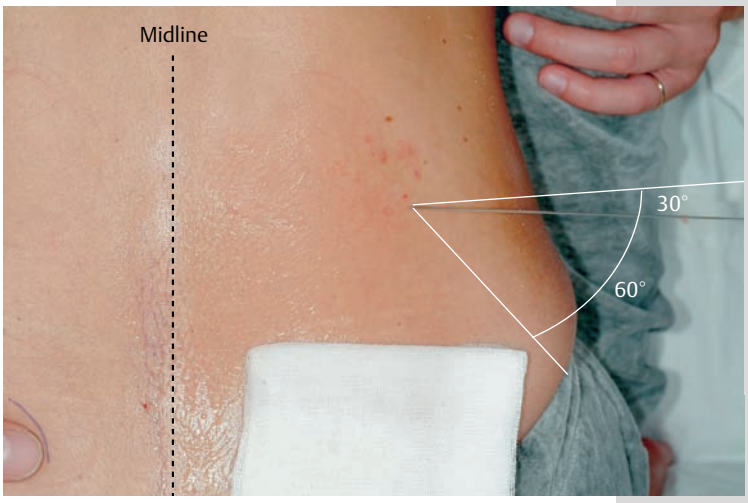


Fig. 9.27 Posterior view of the patient: The needle is positioned vertical to the skin (see **Fig. 9.24**) and then positioned in the horizontal plane at a 60° angle in a lateral direction.

Fig. 9.28 Positioning the needle at 60° using a goniometer, demonstrated on an anatomical specimen.

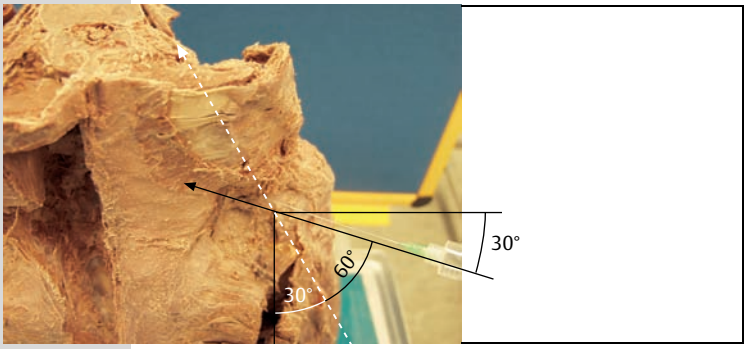


Fig. 9.29 Positioning the needle at 60° without the use of a goniometer, demonstrated on an anatomical specimen. It is clear from this figure that if a smaller angle (e. g., 30–40°) were used, the injection would end up a lot more lateral of the foraminal articular region, increasing the risk of injury to organs. The use of a goniometer is recommended when the practitioner is still learning the technique.

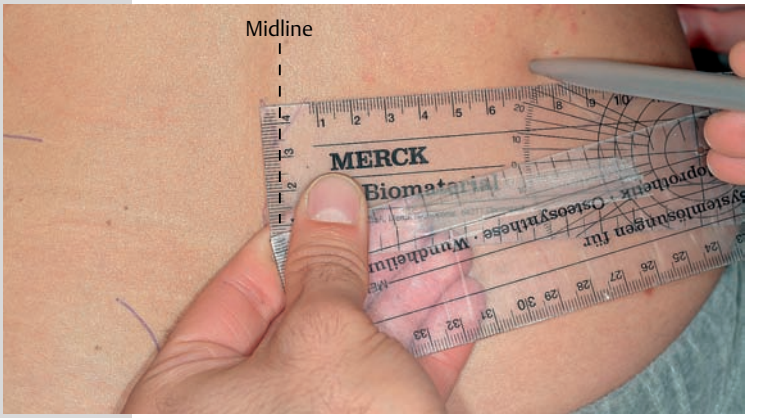


Fig. 9.30 The injection site and position of the needle required for infiltration into the foraminal articular **region of L3/4** and the **L3 nerve root**, demonstrated on a patient. The injection site is located directly above the iliac crest, 8 cm lateral to the midline. The needle is placed at a 60° angle and inserted along the horizontal plane, reaching the foraminal articular **region of L3/L4** and the exiting **L3 nerve root**.

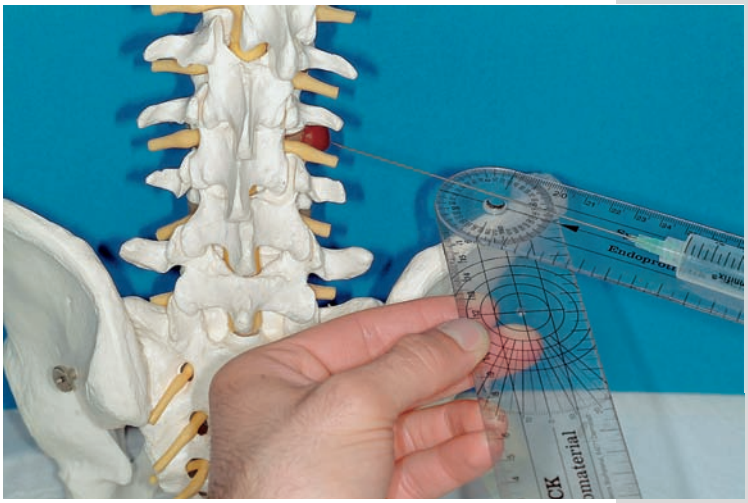


Fig. 9.31 The needle position required for the infiltration of the L3/L4 foraminal articular region and the L3 nerve root, demonstrated on a skeleton.

Fig. 9.32 The needle position required for the infiltration of the L3/L4 foraminal articular region and the L3 nerve root, demonstrated on an anatomical specimen.

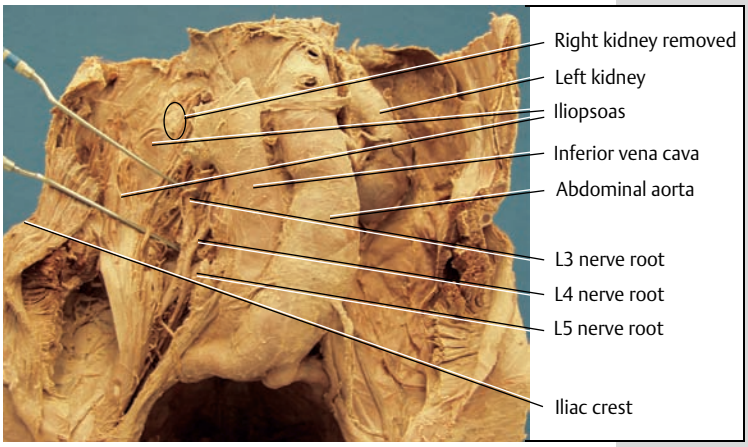


Fig. 9.33 Pelvic specimen, anterior view: Topography of the confluent nerve roots demonstrated on an anatomical specimen from the anterior view. Both dissectors are holding the right iliopsoas muscle to the side.

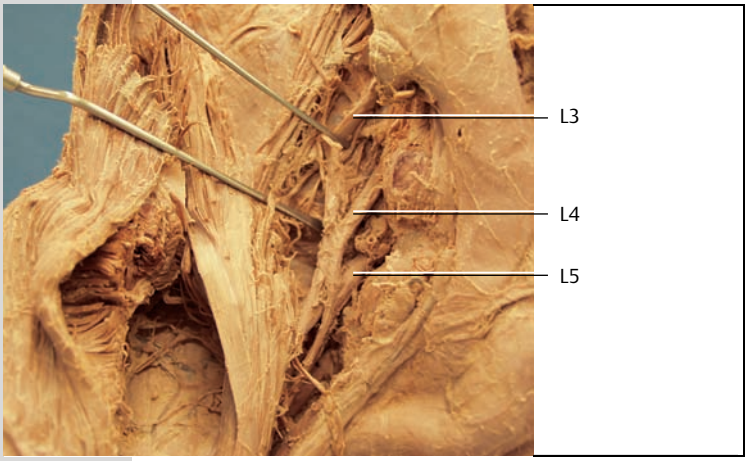


Fig. 9.34 The right confluent L3, L4, L5 nerve roots and their exiting roots.

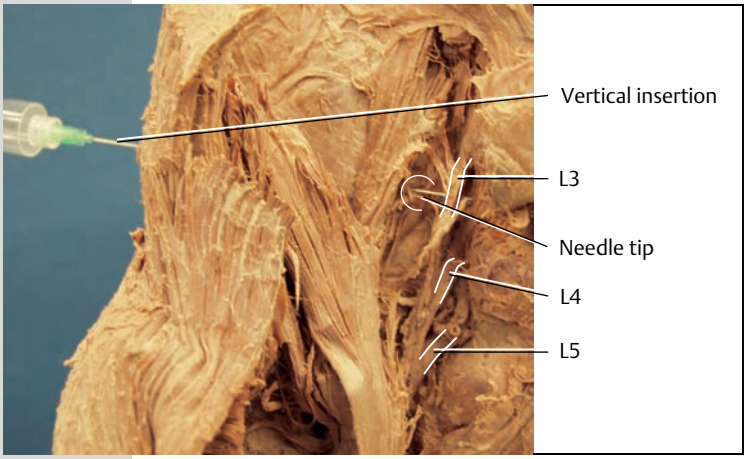


Fig. 9.35 LSPA of the L3/L4 foraminal articular region. The tip of the needle indicates the right **L3 nerve root**. The right L4 and L5 nerve roots are found below this.

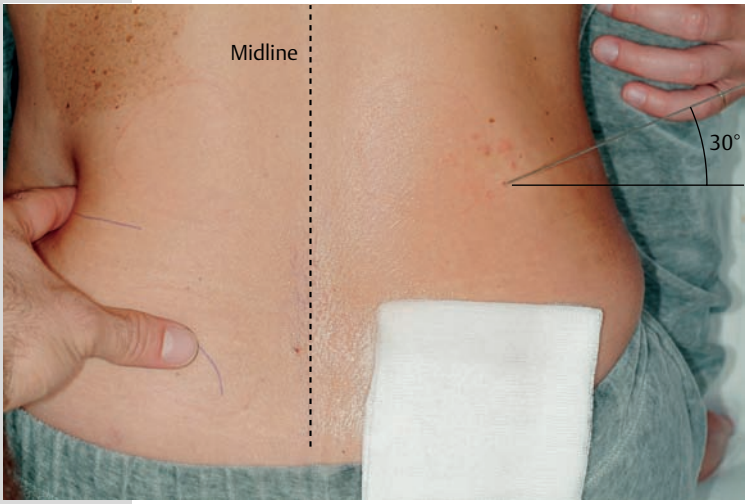


Fig. 9.36 The injection site and position of the needle needed for infiltration into the foraminal articular **region of L4/5** and the **L4 nerve root**, as demonstrated on a patient. The L4/L5 foraminal articular region and the L4 nerve root are reached by raising the needle by ~30°. The needle is inserted superior to the L5 transverse process for 1–2 cm until bony contact is made. The tip of the needle is then positioned on the lateral facet, directly next to the L4/L5 intervertebral foramen or on the side wall of the vertebral body.

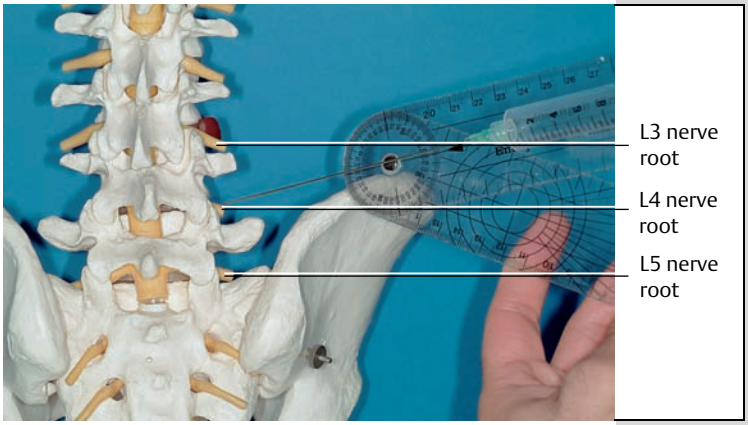


Fig. 9.37 Needle position required for the infiltration of the L4/L5 foraminal articular region, as demonstrated on a skeleton.

Fig. 9.38 Needle position required for the infiltration of the L4/L5 foraminal articular region and the L4 nerve root, as demonstrated on an anatomical specimen. Posterior view.

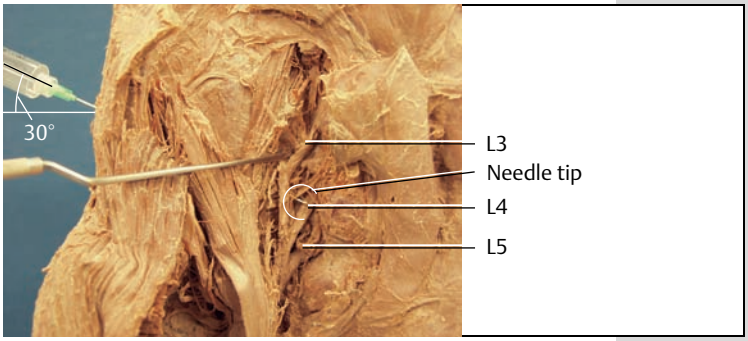


Fig. 9.39 Needle position required for the infiltration of the L4/5 foraminal articular region. The tip of the needle indicates the L4 nerve root, as demonstrated on an anatomical specimen. Anterior view.

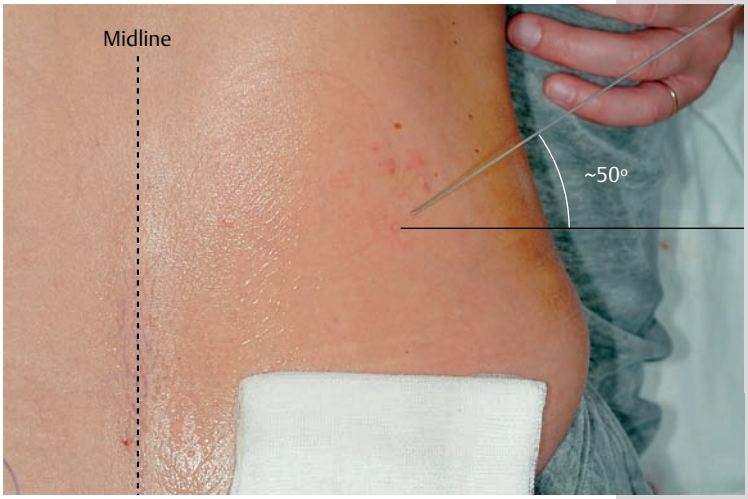


Fig. 9.40 Injection site and needle position required for the infiltration into the foraminal articular **region of L5/S1** and the **L5 nerve root**, demonstrated on a patient (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

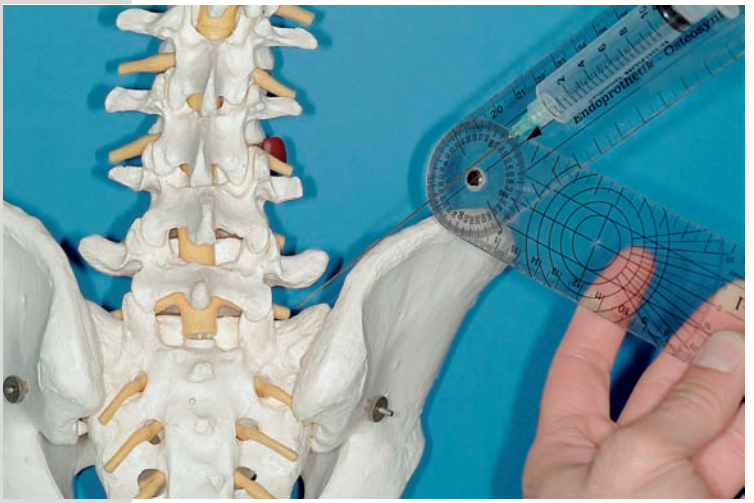


Fig. 9.41 Needle position required for the infiltration of the L5/S1 foraminal articular region, demonstrated on a skeleton. Locating the L5/S1 foraminal articular region. The needle tip is pointing to the exiting L5 nerve root and the right traversing S1 nerve root.

Fig. 9.42 Needle position required for the infiltration of the L5/S1 foraminal articular region, demonstrated on an anatomical specimen. Posterior view.

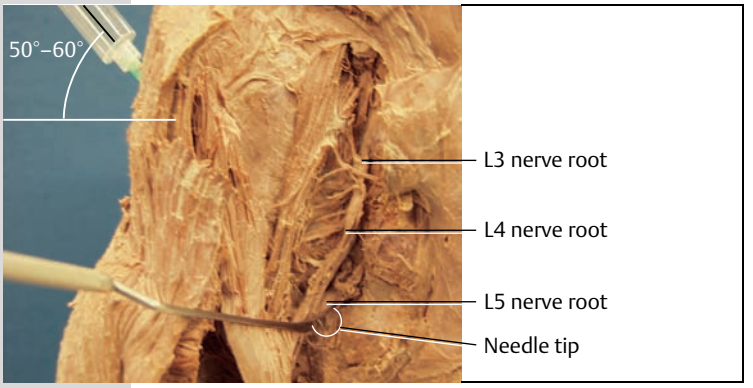


Fig. 9.43 Needle position demonstrated on an anatomical specimen; anterior view. The tip of the needle points toward the foraminal articular **region of L5/S1**. The right exiting **L5 nerve root** can be seen in this figure.

Fig. 9.44 Administering the LSPA. The physician and the assistant continually monitor the patient verbally and by touch.

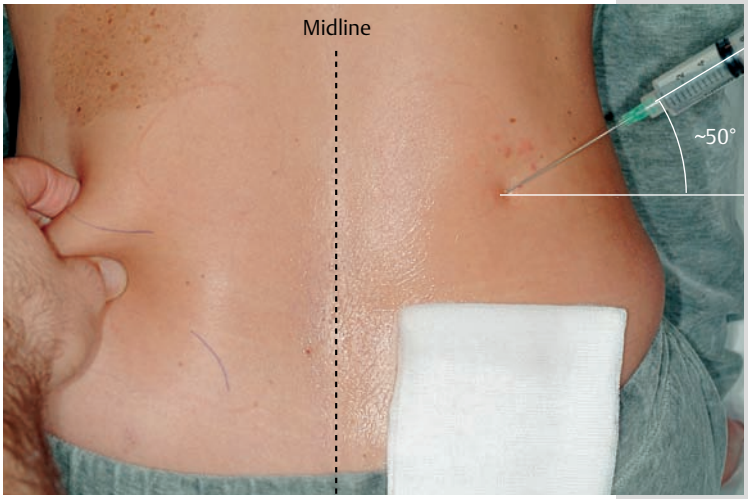


Fig. 9.45 Conducting the LSPA into the L5/S1 foraminal articular region and the L5 nerve root, demonstrated on a patient. The L5/S1 foraminal articular region, the exiting L5 nerve root, and the traversing S1 nerve root located more medially are located between the lower edge of the L5 transverse process and the upper edge of the sacrum. This region is reached by raising the needle in the vertical plane to an angle of ~50–60°. The patient is distracted by pinching the skin on the contralateral side (gate control) when the skin is punctured and during the final phases of the LSPA. The patient is informed when the needle will puncture the skin ("You are now going to feel a pin prick"). The skin resistance should then be overcome as soon as possible. Pain due to the injection itself can be prevented by injecting and further inserting the needle (with aspiration) simultaneously until bony contact is made with the foraminal articular region. Pre-injection also prevents painful nerve root contact. Direct nerve root contact tends to be rare when an angled needle position is used, because part of the transverse process near the pedicle covers the exiting nerve root (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

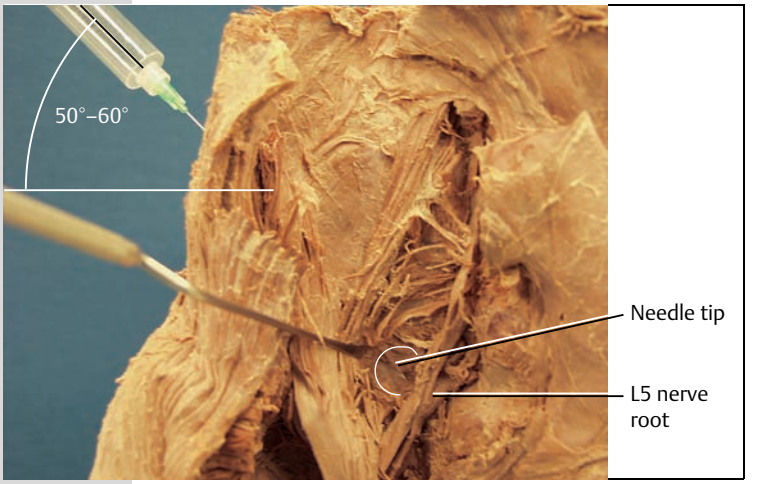


Fig. 9.46 Needle position demonstrated on an anatomical specimen; anterior view. The tip of the needle points toward the L5/S1 foraminal articular region. The right exiting L5 nerve root can be seen on this figure.

Fig. 9.47 At the conclusion of the LSPA, a nonallergenic adhesive dressing is placed over the injection site and should be removed after an hour. The needle injection site should be treated with care and inspected daily, as daily lumbar spinal nerve analgesias are planned within the scope of the minimally invasive spinal therapy for severe pain.

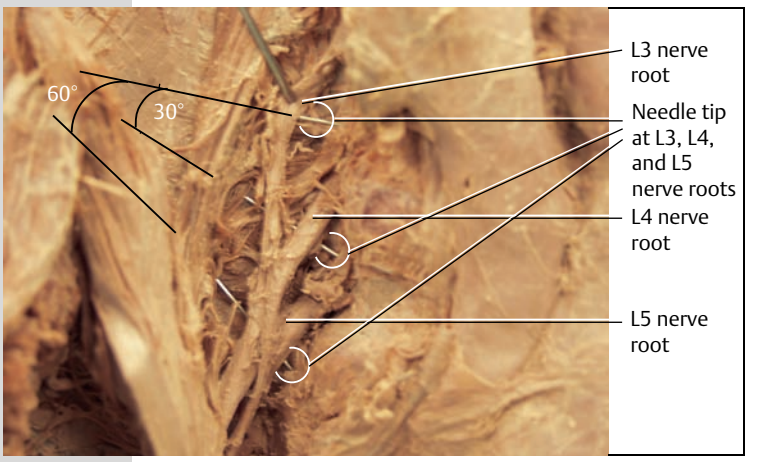


Fig. 9.48 Review of needle position during the LSPA for the right L3/L4, L4/L5, and L5/S1 foraminal articular region and the different angles required. Demonstrated on an anatomical specimen; anterior view.

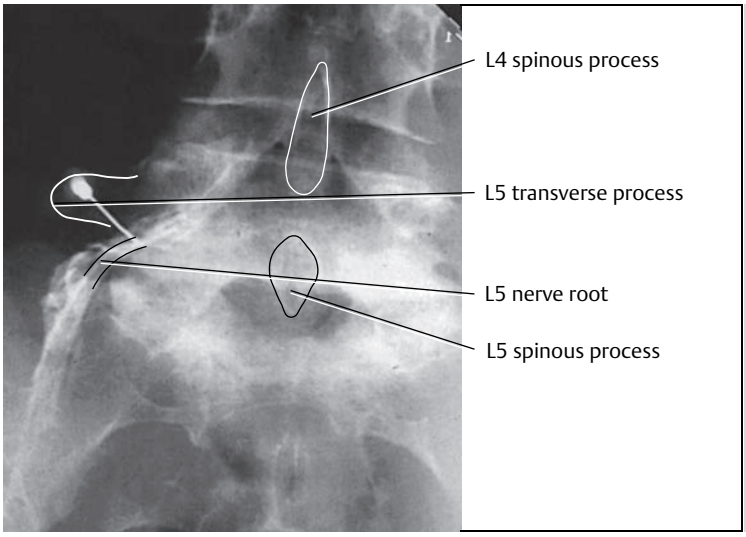


Fig. 9.49 Radiculography of the L5 nerve root. This is used as a diagnostic measure, to identify a compromised nerve root, and in pain therapy, when a nerve root compression syndrome is present in the intervertebral foramen.

Radiculography

Principle

Imaging and paravertebral analgesia of the spinal nerve root directly at the point where it exits the intervertebral foramen.

Indication

This type of local injection is administered only once. It is used both as a **diagnostic** local injection to identify the affected nerve root and also to **differentiate the indications for surgical intervention**, ascertaining whether root decompression should be considered in addition to fusion.

Macnab and Dali (1971) originally intended this form of perineural infiltration to be used for diagnosis. Van Akkerveeken (1989) later used it additionally for therapeutic purposes.

Technique

The patient lies prone with a cushion under the abdomen, flattening out the lordotic curvature of the lumbar spine. Under image monitoring in the operating theater, a long, thin puncture needle is inserted until it reaches the L5 transverse process and is then further inserted until it reaches the nerve root above (L4) or below (L5). Once the typical radiating pain (memory pain) is triggered, the perineural tube is filled with a contrast medium to assess the position of the needle and to verify the correct nerve root level. After this, the needle is slightly retracted and, following aspiration, 2–5 mL of local anesthetic is infiltrated perineurally, to eliminate the pain that has previously been induced. If surgery is not scheduled to follow immediately, 10 mg of cortisone crystal suspension can also be injected (**Fig. 9.49**).

Lumbar Facet Infiltration

Principle

Switching off nociceptors in the lumbar zygapophyseal joint capsule using temporary blocks with a local anesthetic and an additional steroid, when necessary.

Indication

Symptoms originating in the zygapophyseal joint, i. e., facet syndromes, hyperlordosis-related low back pain, pseudoradicular syndromes.

Technique

The patient either sits or lies prone with a cushion under the abdomen, flattening out the lordotic curvature of the lumbar spine. The zygapophyseal joint capsule is reached by vertically inserting a thin 6–8 cm cannula; the insertion site is 2–2.5 cm paravertebral, between the spinous processes. Patients will experience their typical radiating pain when the tip of the cannula intrudes into the joint or the joint capsule. Intra-articular positioning of the needle is not required. A periarticular–pericapsular infiltration generally suffices.

The inferior 4–6 lumbar zygapophyseal joints are usually infiltrated at the same time: 2 mL of local anesthetic combined with a cortisone crystal suspension is used for each joint. Depot local anesthetics are used alone when injections are administered repeatedly at short intervals. Therapy with the patient in the flexed posture is always used in conjunction with facet infiltrations. The aim is to flatten out the lumbar lordosis, which can be achieved by placing the patient in the Fowler position, conducting exercises that start in a pain-relieving position, and using flexion orthoses.

After some practice, it is not necessary to use radiographic image monitoring for this technique. It is possible to carry out facet infiltration using concurrent sonographic monitoring to reach the dorsal zygapophyseal joint complex (Grifka et al. 1999).

Lumbar Facet Infiltration Procedure (Figs. 9.50–9.75)

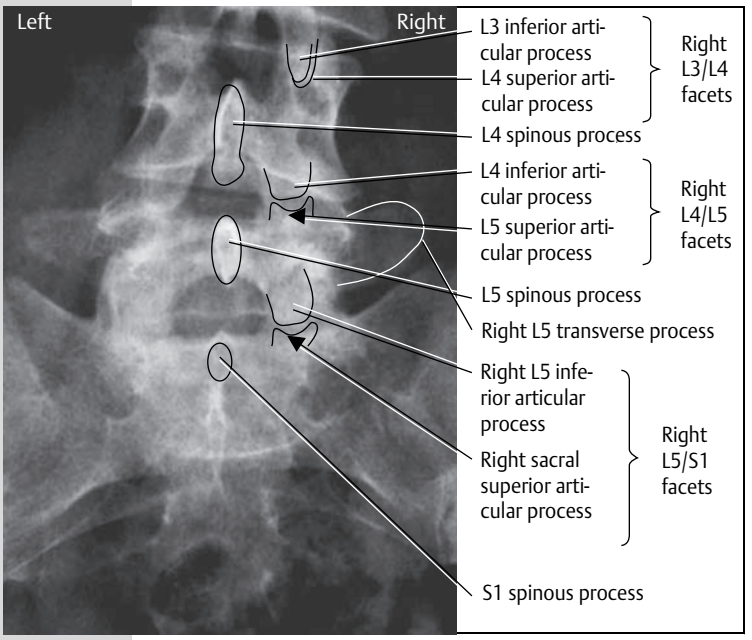


Fig. 9.50 An anteroposterior image of the lumbar spine is required for facet infiltration, where infiltration follows the palpation of anatomical orientation points. It should be displayed the right way around (right = right). The physician looks at the image while simultaneously viewing the sitting patient's back. Note any scolioses, anomalies, transitional vertebrae, and asymmetrically positioned facets.

Fig. 9.51 View of the inferior lumbar region from the sitting practitioner's point of view. The patient's back is free of clothing so that the inferior costal arch, the waist, the iliac crests, and the sacroiliac region (SIJ) can be easily assessed and palpated. The skin must be intact and show no signs of infection.

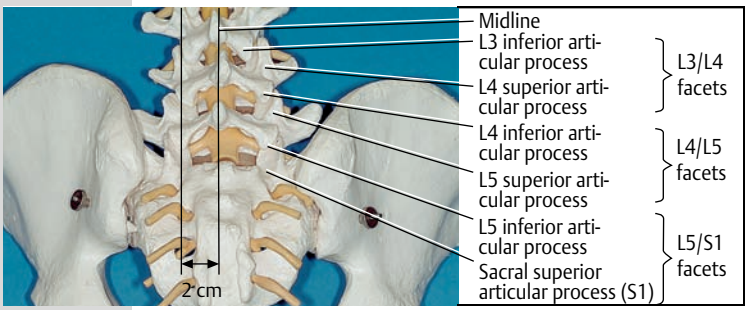


Fig. 9.52 Inferior skeletal lumbar spine, posterior view. The bony sections of the L3/L4, L4/L5, and L5/S1 zygapophyseal joints are relatively wide and can reliably be found 2–2.5 cm lateral to the midline. The joint capsule is infiltrated. The capsule acts as a hood, lying over the joint.

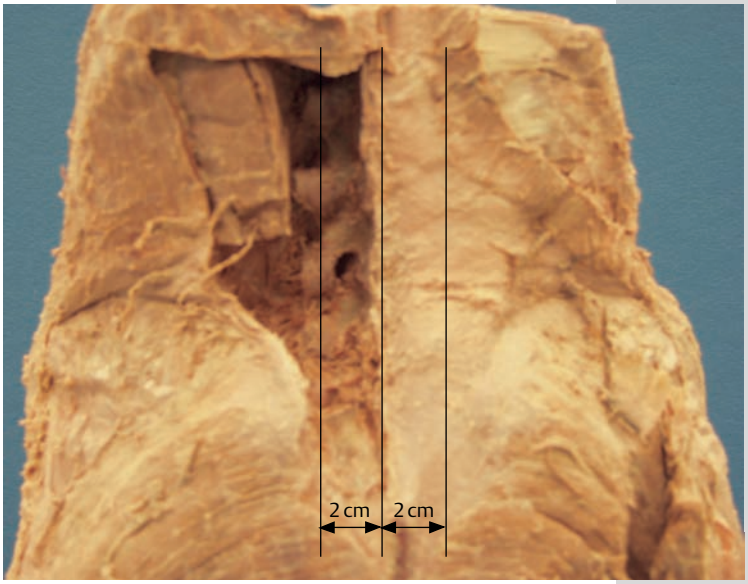


Fig. 9.53 Topography of the posterior section of the vertebral motor segment, displayed on a pelvic specimen. Posterior view: The muscles have been removed on the left-hand side. The L3/L4, L4/L5 and L5/S1 facet complexes are each found between the spinous processes, ~2–2.5 cm lateral.

Fig. 9.54 The physician is positioned directly behind the patient who is sitting on a higher examination couch. It should be comfortable to palpate the iliac crests. The pulse oximeter is placed next to the patient. Bimanual palpation and orientation on the sitting patient. The thumbs slide medially from a lateral position, over the posterior iliac spine, and in the sulcus between the iliac spine and the medial sacral crest. The index and middle fingers palpate the iliac crest (from Theodoridis T, Ludwig J, and Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

Fig. 9.55 Bimanual palpation of both posterior superior iliac spines and the iliac crests, demonstrated on a skeleton.

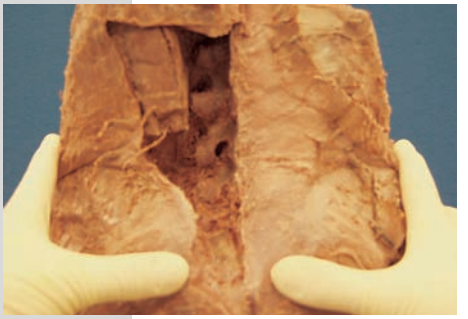


Fig. 9.56 Bimanual palpation of both posterior superior iliac spines and the iliac crests, demonstrated on an anatomical specimen.

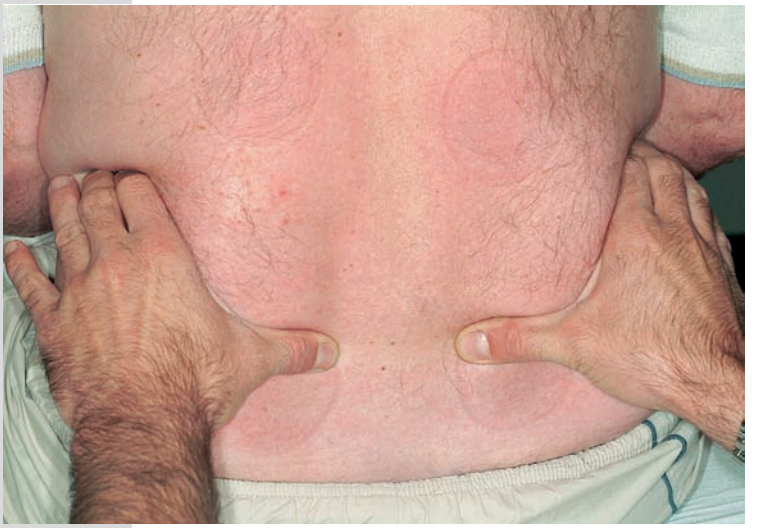


Fig. 9.57 Bimanual palpation of both posterior superior iliac spines and the iliac crests, demonstrated on a patient.

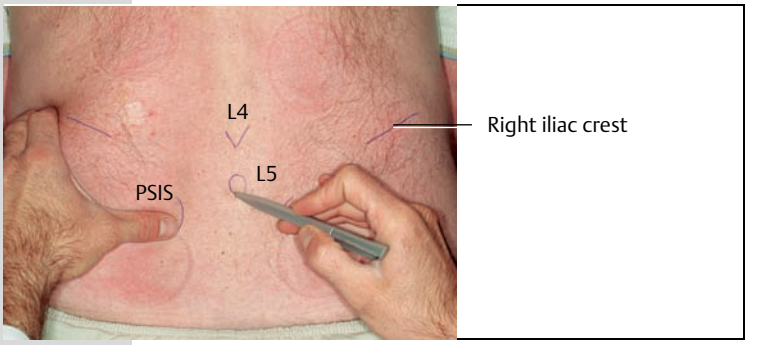


Fig. 9.58 Marking the tips of the L4 and L5 spinous processes in relation to the iliac crest. The connecting line between the iliac crests corresponds to the height of the L4 spinous process (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany:

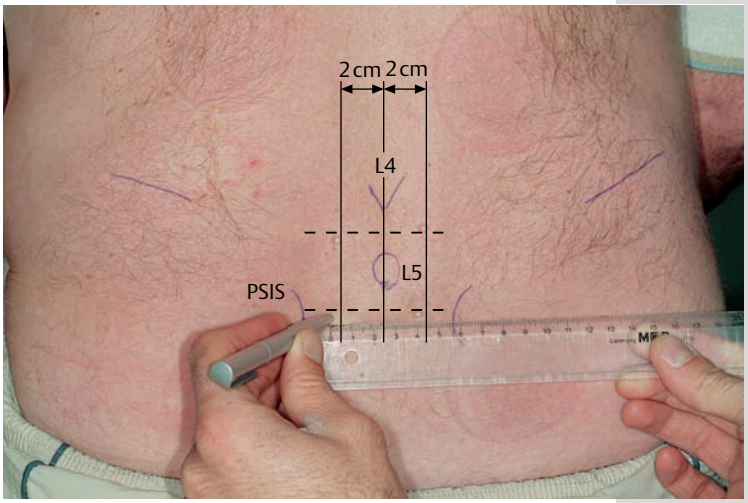


Fig. 9.59 Injection sites for the L4/L5 and L5/S1 facet infiltration are found between the spinous processes, ~2 cm lateral to the midline, and are marked by rotating a pen (with ink cartridge retracted) over the sites. The location of the mark remains visible, even after disinfection.

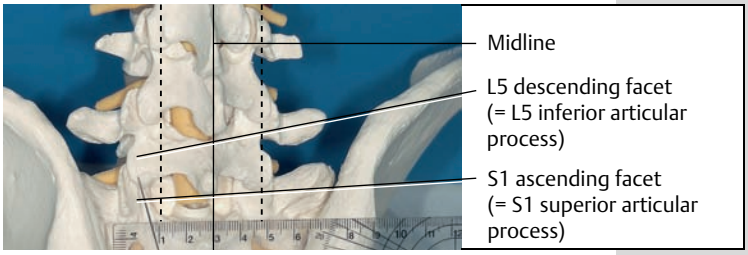


Fig. 9.60 Needle position required for the L5/S1 facet joint capsule, shown on a skeleton.

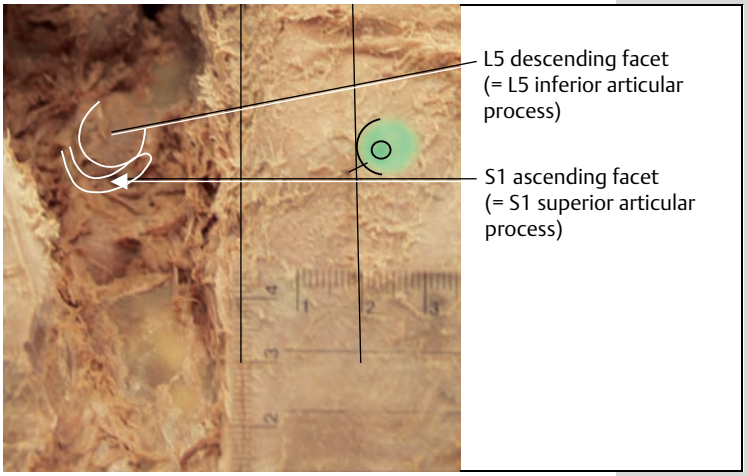


Fig. 9.61 Needle position at L5/S1, demonstrated on an anatomical specimen on the right. The descending L5 facet and the ascending S1 facet are marked on the left. In this case, the facet complex's midpoint is found 2 cm lateral to the midline.

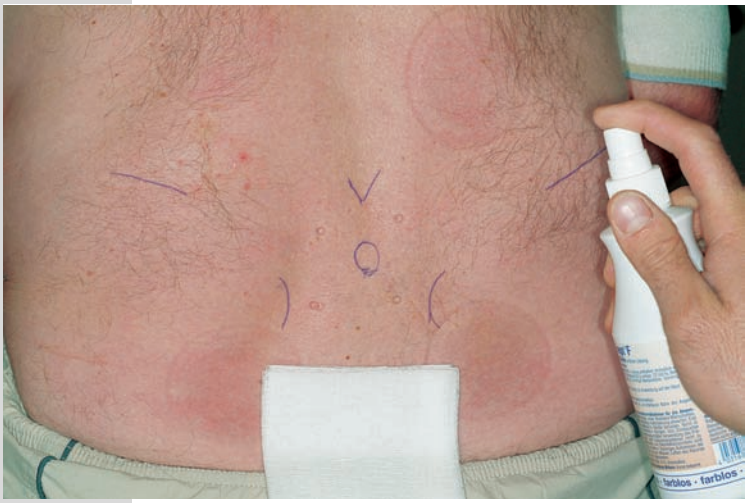


Fig. 9.62 Towels are used to protect the patient's clothing. The disinfection process can then begin.

Fig. 9.63 Ink markings may be removed when the skin is disinfected, but the marked injection sites for the bilateral facet infiltration at L4/L5 and L5/S1 remain.

Fig. 9.64 Right L4/L5 facet infiltration. The needle is inserted vertical to the skin surface at the marked L4/L5 site on the right.

Fig. 9.65 Both hands are used to guide the injection system. The left hand rests on the patient, if necessary. The needle is inserted until bony contact is made. Fan-shaped infiltration of ~2.5 mL local anesthetic into the posterior capsule of the zygapophyseal joint when bony contact is made and following aspiration. Both hands are used to guide the syringe system (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

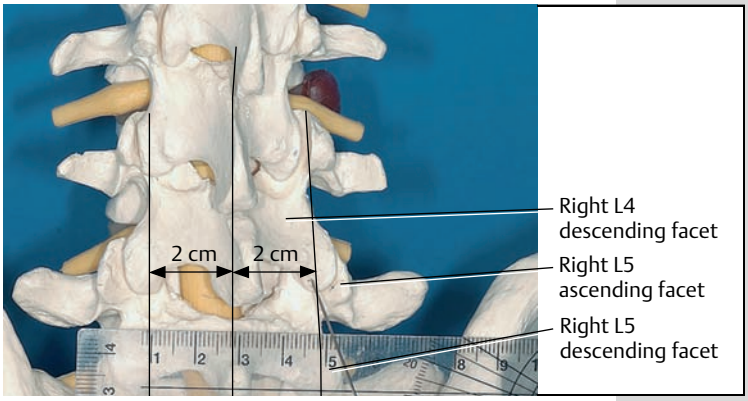


Fig. 9.66 Bony contact with the right L4/L5 facet complex, demonstrated on a skeleton. The facet complex is found 2 cm lateral of the midline. Further insertion is not possible.

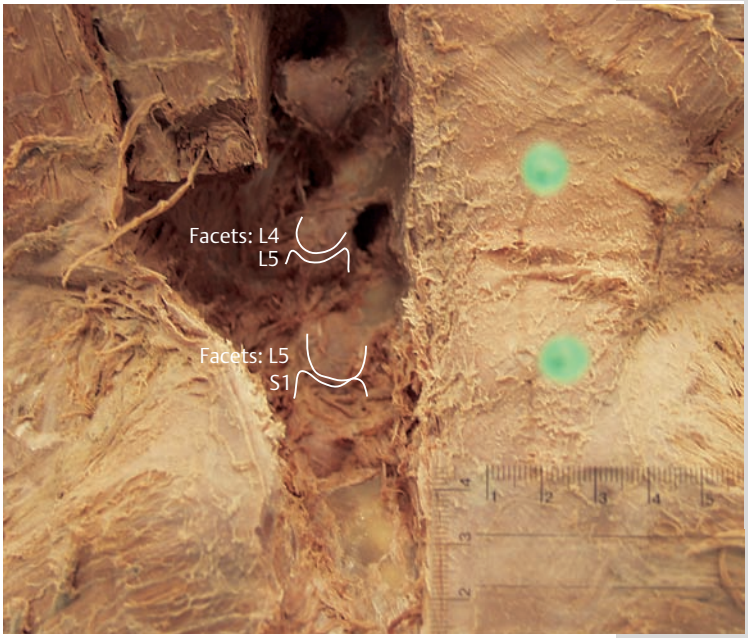
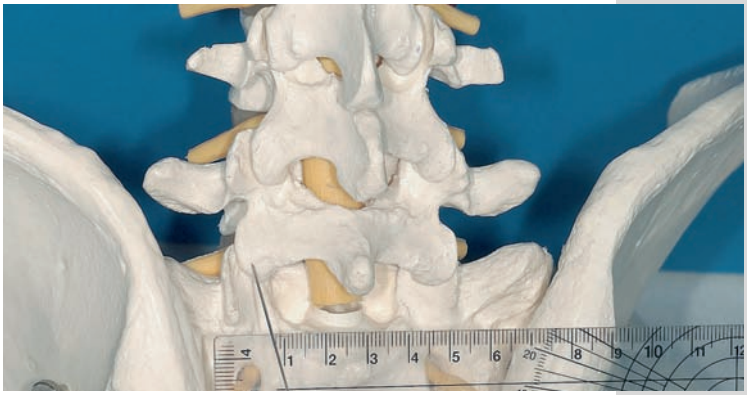


Fig. 9.67 Facet infiltration at L4/L5 and L5/S1 on the right, demonstrated on an anatomical specimen. The corresponding posterior sections of the facet have been dissected on the left hand side.

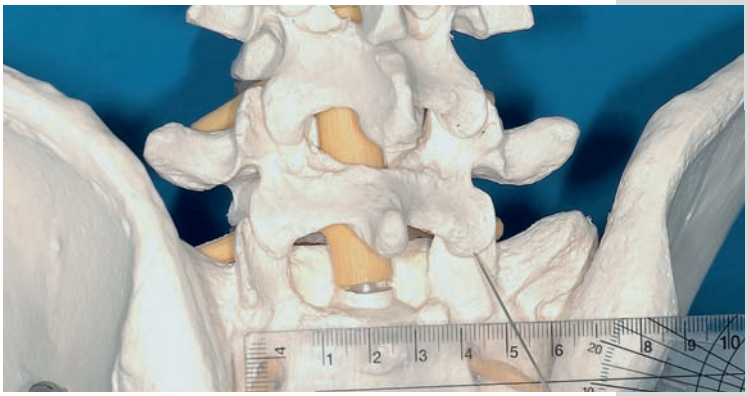
Fig. 9.68 Left L4/L5 facet infiltration. The needle is inserted vertical to the skin surface at the marked L4/L5 site on the left.

Fig. 9.69 Needle position required for the left L4/L5 facet joint capsule, demonstrated on a skeleton.

Fig. 9.70 The needle is inserted until bony contact is made. Injection is permitted only when bony contact has been established. A further 2.5 mL of local anesthetic is infiltrated into the posterior section of the capsule of the left L4/L5 facet complex.



a



b

Fig. 9.71a, b The procedures for the left and right L5/S1 facet complexes respectively, demonstrated here on a skeleton.

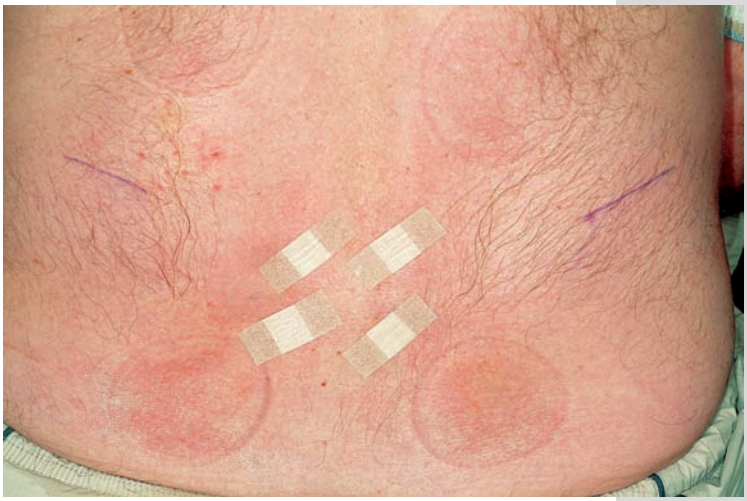


Fig. 9.72 A nonallergenic adhesive dressing is placed over the injection sites and removed after an hour. No skin reaction should be seen at the injection sites.

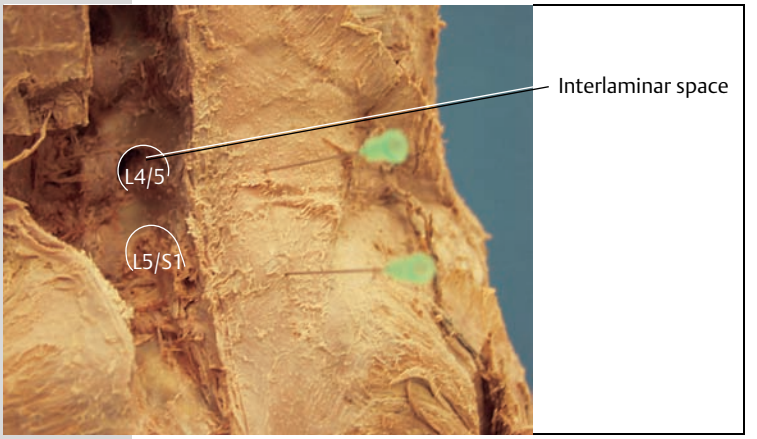


Fig. 9.73 Facet capsule infiltration at L4/L5 and L5/S1 on the right, demonstrated on an anatomical specimen. Left/lateral view. The L4/L5 and L5/S1 facet complexes have been dissected on the left-hand side. It is absolute necessary that bony contact is established. The midpoint of the facet complex is found 2 cm lateral to the midline. The interlaminar space is found further medial and the exiting nerve root more lateral and somewhat deeper. The ideal point for facet capsule infiltration is found at the transition between the descending and ascending facets by examining the interspinous area 2 cm lateral without injecting.

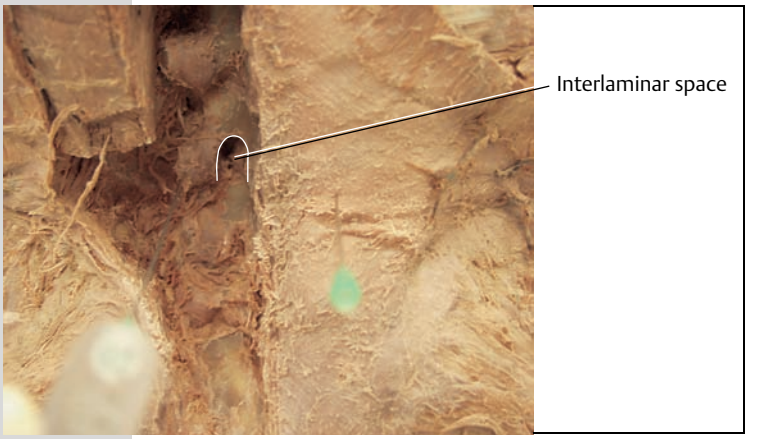


Fig. 9.74 Right L4/L5 facet infiltration, demonstrated on an anatomical specimen. The exposed L4/L5 joint capsule is shown on the left. The injection site is significantly lateral to the lateral border of the interlaminar space.

Fig. 9.75 Facet infiltration is recommended when the spinal canal stenosis decompensation (triangles) is initiated by the activation of arthrosis in the zygapophyseal joint. This injection can also be conducted under radiographic or CT monitoring when joint effusion (arrow) is distinct. The joint effusion is shown on the MRI as lucency with an accumulation of fluid (Krämer and Köster 2001, Krämer et al. 2004).

Ligament Infiltration at the Sacroiliac Joint (SIJ Block)

Principle

Switching off irritated nociceptors at the transitional points between ligament and bone in the posterior ligamentous system of the sacroiliac joint and at the sites of insertion for the iliolumbar ligament.

Indication

1 Sacroiliac joint (SIJ) blockade
2 Local lumbar syndrome
3 SIJ syndrome
4 Pseudoradicular lumbar syndrome
5 Complementing manual therapy
6 Sacroiliitis.

Technique

The dorsal ligamentous system in the sacroiliac joint area is best reached by inserting the needle at the same level as the ipsilateral posterior superior iliac spine (PSIS) and the S1 spinous process, and exactly along the midline between them. This spot is marked and the needle is then inserted at a lateral angle of approximately 45° to the skin surface.

The local anesthetic diffuses sufficiently into the entire dorsal ligamentous system of the sacroiliac joint when the syringe is inserted and retracted and the needle makes ligamentous or bony contact.

Effect

When the ligamentous system of the sacroiliac joint is permanently irritated by traction or pressure, which may happen during the blockade process, the nociceptors found at the transitional point between the ligament and bone are activated. This often leads to local or pseudoradicular spinal or SIJ syndromes. Although the ligamentous infiltration described above should not cause major problems in the SIJ area, **intra-articular injection** into the sacroiliac joint is more problematic and is best administered **using image guidance**. This injection is, however, subject to particularly **strict indication guidelines** as ligamentous periarticular infiltration often proves to be sufficient, particularly in the SIJ area.

Sacroiliac Joint Block Injection Procedure (Figs. 9.76–9.97).

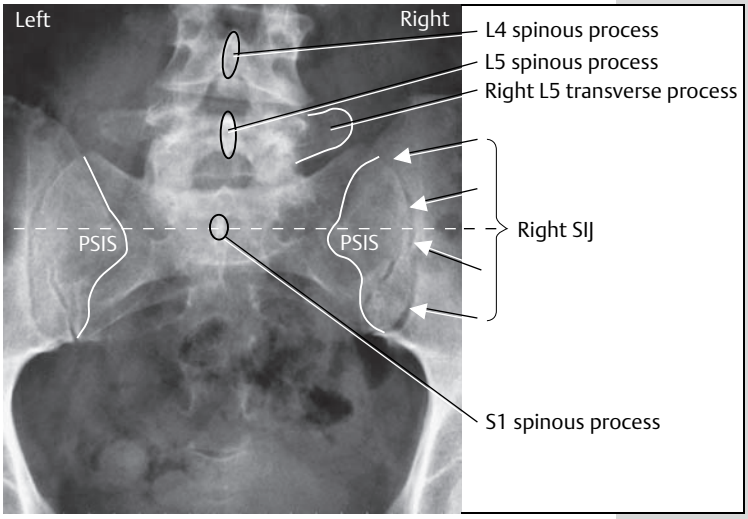


Fig. 9.76 An anteroposterior radiogram of the inferior lumbar spine and the sacroiliac joints should be available when infiltrating the sacroiliac joints. The radiograph is displayed the right way round (right = right) in the field of vision of the practitioner who assesses and palpates the sitting patient's inferior lumbar region from a posterior direction. The radiograph is used to exclude specific diseases in this area and for orientation purposes during the injection.

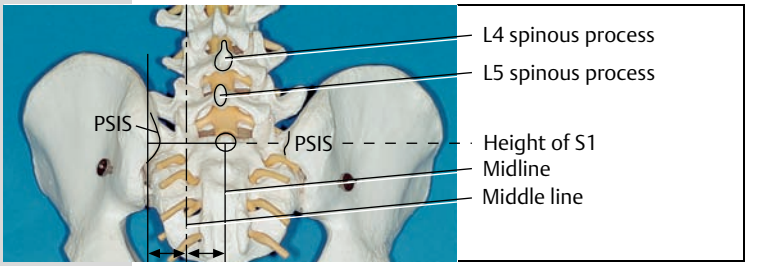


Fig. 9.77 The most important anatomical orientation points, demonstrated on a skeleton: The iliac crest, PSIS, and spinous process are always palpable on slightly flexed sitting patients, even on obese patients.

Fig. 9.78 The examiner is positioned directly behind the patient, who is sitting on a higher examination couch. The examiner must be comfortably able to palpate the iliac crests and the border of the sacroiliac joints bimanually. This arrangement is used for the SIJ block.

Fig. 9.79 Bimanual palpation of the posterior superior iliac spines and the iliac crests, demonstrated on a skeleton.

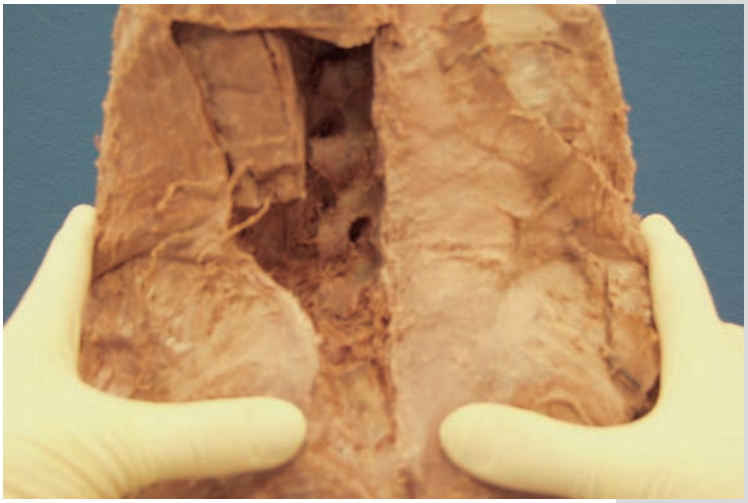


Fig. 9.80 Bimanual palpation of the posterior superior iliac spines and the iliac crests, demonstrated on an anatomical specimen.

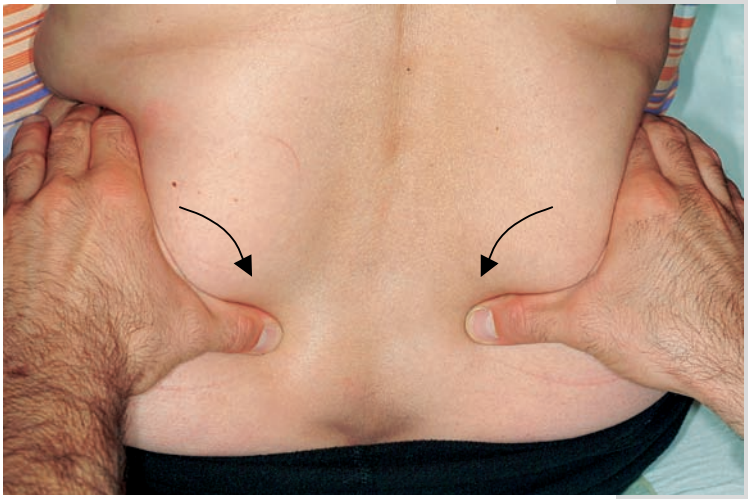


Fig. 9.81 Bimanual palpation and orientation on the sitting patient. The thumbs slide medially from a lateral position, over the posterior iliac spine, and in the sulcus between the iliac spine and the medial sacral crest. The index and middle fingers palpate the iliac crest.

Fig. 9.82 Marking the iliac crests and the posterior superior iliac spines bilaterally.

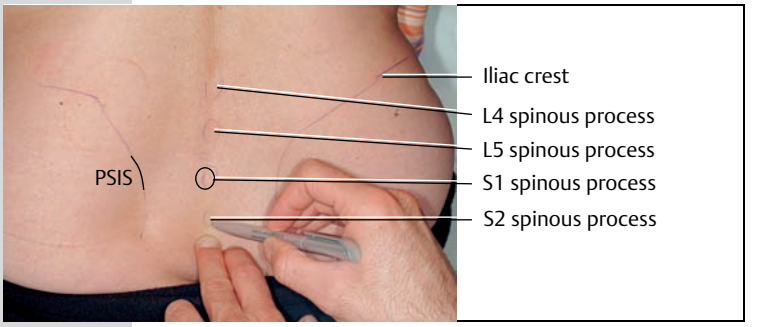


Fig. 9.83 The spinous processes, L4, L5, and the sacrum are additionally marked.

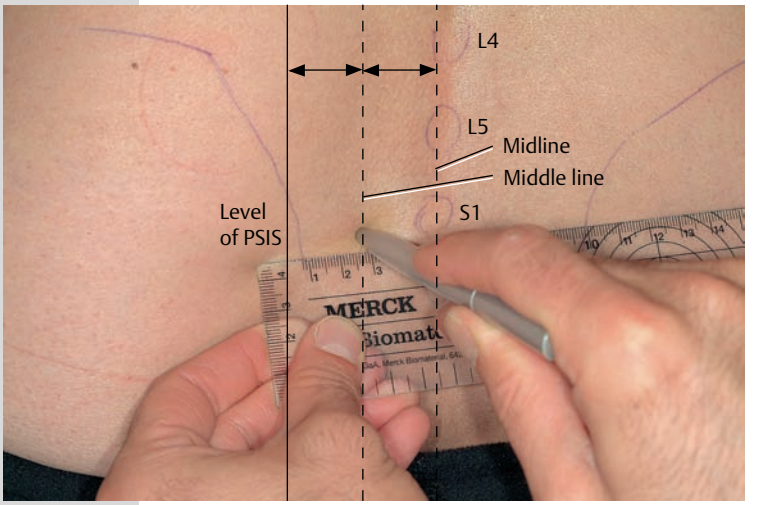


Fig. 9.84 Marking the injection site with a pen (with retracted ink cartridge) exactly in the midline at the height of the ipsilateral posterior superior iliac spine (PSIS) and the S1 spinous process.

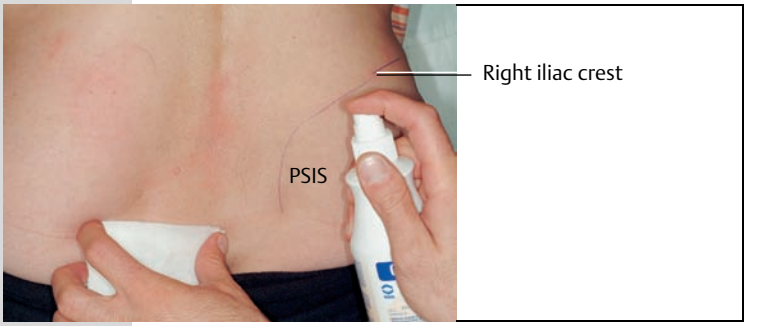


Fig. 9.85 Towels are used to protect the patient's clothing. The disinfection process can then begin. Ink marks may be removed during the skin disinfection, but the pressure mark on the skin marking the injection site for the left SIJ block remains.

Fig. 9.86 The patient sits in a slightly flexed posture for the injection.

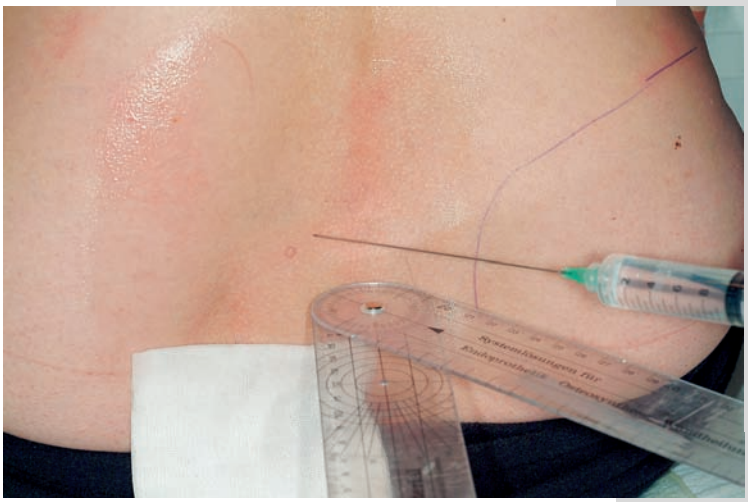


Fig. 9.87 Placing the needle at 45° in the horizontal plane on the patient at the pressure-marked point.

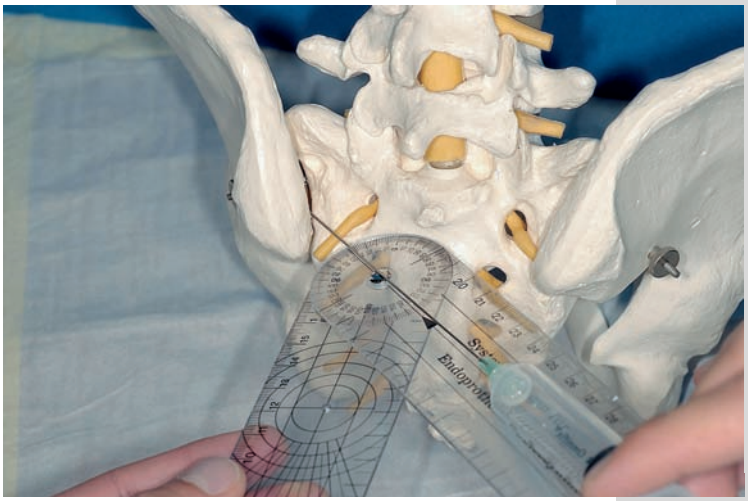


Fig. 9.88 Needle position, demonstrated on a skeleton. The 45° angle of the needle directly targets the middle section of the SIJ.

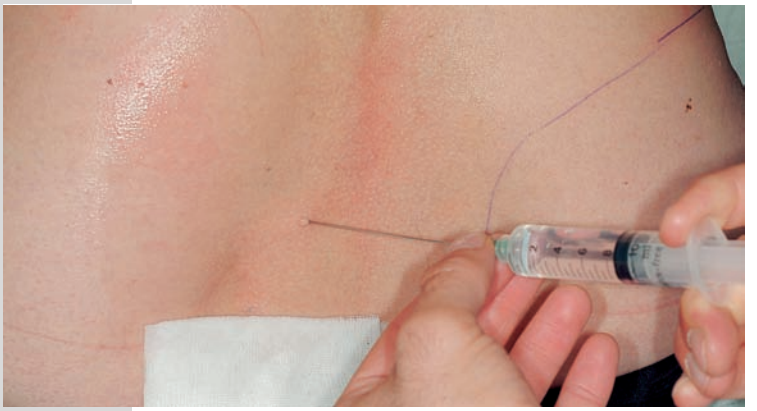


Fig. 9.89 The needle penetrates the skin quickly and is then further inserted until it makes bony contact with the posterior section of the SIJ. Both hands are used to guide the injection system in searching for the SIJ gap.

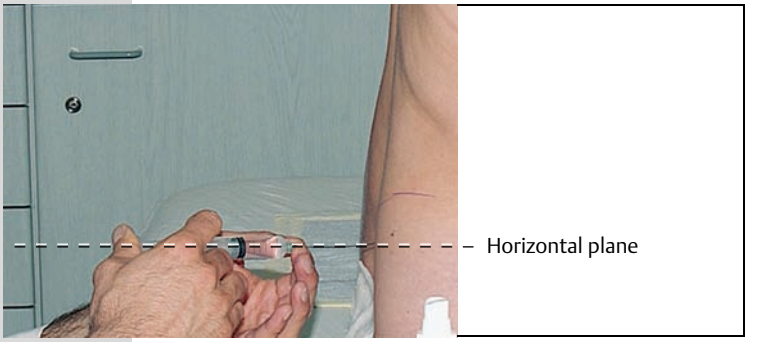


Fig. 9.90 Side view during the SIJ infiltration. The needle is predominantly located in the horizontal plane. Approximately 3 mL of local anesthetic is infiltrated in a fan shape.

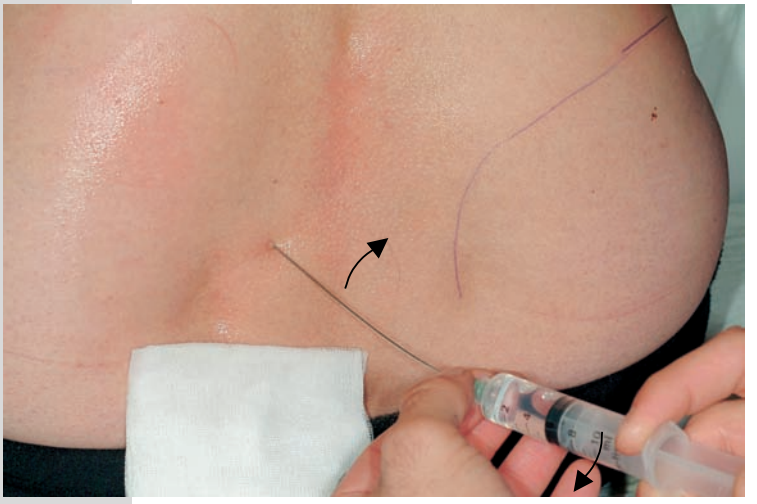


Fig. 9.91 The syringe system is then retracted. Using both hands, the syringe is lowered and the needle is raised at the same time and inserted once more until bony/capsule contact is made. The superior section of the SIJ is reached using this method. A further 3 mL of local anesthetic is infiltrated in a fan shape.

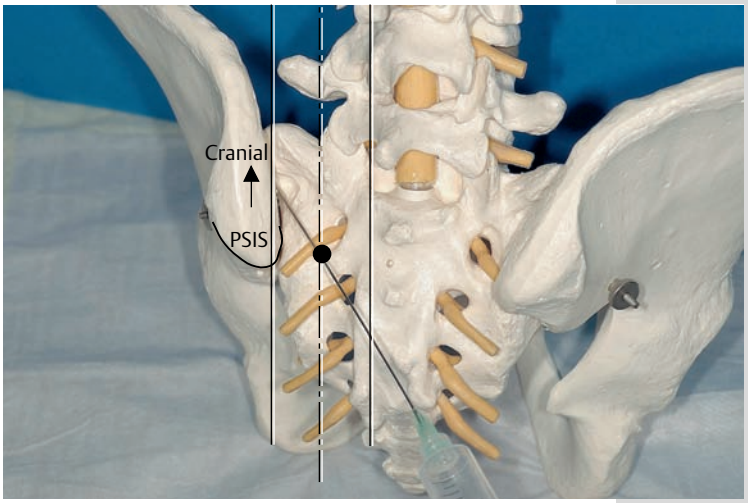


Fig. 9.92 Infiltration of the superior section of the SIJ, demonstrated on a skeleton.

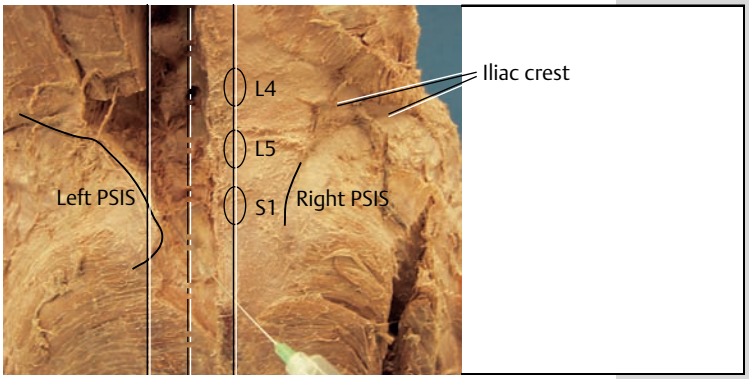


Fig. 9.93 Infiltration of the superior section of the SIJ, demonstrated on an anatomical specimen.

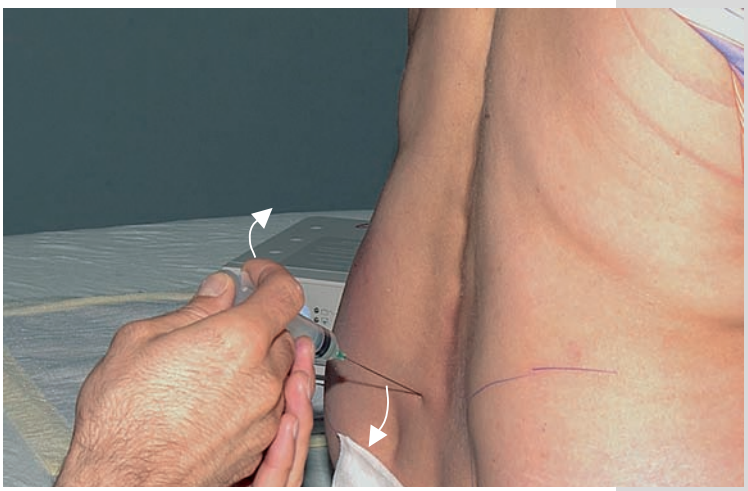


Fig. 9.94 The syringe system is then retracted again, the syringe is raised and the needle is lowered at the same time using both hands, and inserted once more until bony/capsule contact is made. The inferior section of the SIJ is reached using this method. The remaining local anesthetic is infiltrated in a fan shape.

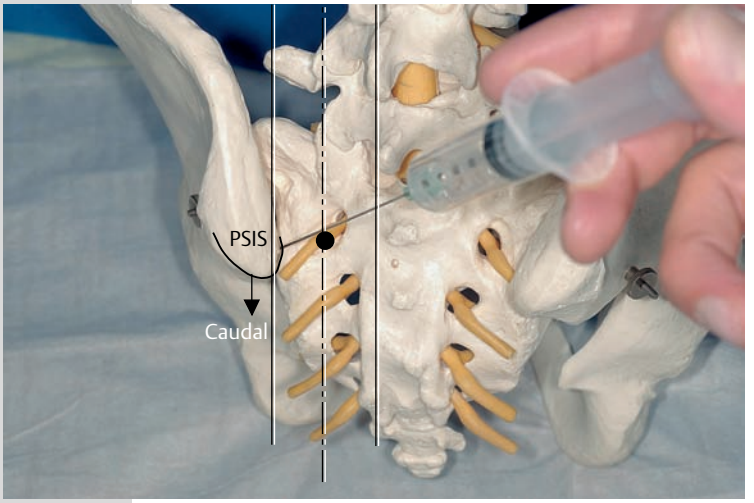


Fig. 9.95 Infiltration of the inferior section of the SIJ, demonstrated on a skeleton.

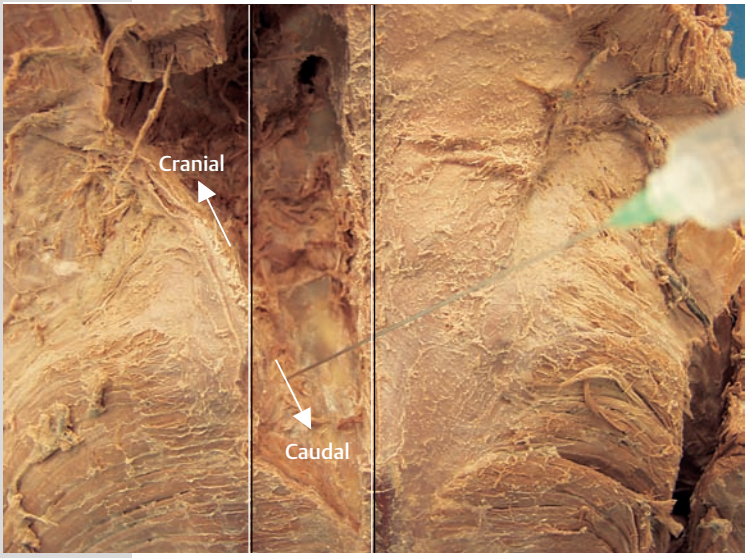


Fig. 9.96 Infiltration of the inferior section of the SIJ, demonstrated on an anatomical specimen. The soft tissue has been removed on the left-hand side. The SIJ is bordered medially and laterally by the sacrum and coccyx over a broad area. There is therefore no need to worry about inserting the needle laterally into the soft tissue and medially into the vertebral canal.

Fig. 9.97 Nonallergenic adhesive dressing, which should be removed after an hour.

Lumbar Epidural Pain Therapy

There are many ways to reach the lumbar epidural space using a cannula.

Interlaminar access is used by anesthesiologists to insert peridural catheters for lumbar, spinal, and peridural anesthesia. Apart from the errors and dangers that arise from prolonged use of catheters (Donner 1995), its main disadvantage in orthopedic pain treatment is that catheterized patients cannot participate in many of the interventions available in the accompanying physiotherapy program. For this reason single epidural injections using the so-called "single shot" technique are employed in orthopedic pain therapy. Interlaminar access is commonly used for conventional epidural injections using the loss-of-resistance technique, or for epidural perineural injections using the new two-needle technique for the anterior epidural space.

Access via the **sacral hiatus** is very common in pain therapy and is used especially for inferior lumbar root syndromes (Bush and Hillier 1991).

It is also possible to reach the lumbar epidural space indirectly **via the intervertebral disk**, by a perforated anulus fibrosus posteriorly. We take advantage of this possibility when discography shows that contrast agent is flowing into the epidural space and the planned chemonucleolysis is not a viable option. In these cases, the cortisone is administered into the intervertebral disk and flows out of the disk directly into the anterior epidural space, the area where the nerve root is compromised.

Access to the lumbar epidural space via the **intervertebral foramen** is only possible with CT monitoring. This route is reserved for special cases, such as the therapy-resistant postdiscotomy syndromes with compression of the exiting nerve root (**Table 9.8**).

Table 9.8 Access Routes into the Lumbar Epidural Space Used in Pain Therapy

Access	*Injection*
Interlaminar	1 Posterior epidural 2. Epidural perineural
Sacral hiatus	Sacral epidural
Intervertebral disk	Intradiscal
Intervertebral foramen	Intraforaminal

Sacral Epidural Injection

Principle

Injection into the lumbar epidural space through the sacral hiatus.

This approach is used, for example, to infiltrate the inferior section of the sacral plexus with a local anesthetic. The resulting insensitivity is generally restricted to the S3–S5 nerve roots along the lines of a saddle block anesthesia. The injected substance, e. g., a saline cortisone crystal suspension, can also travel more superiorly when the pelvis is elevated. In this case, the substance acts in the peridural space of the inferior lumbar spine.

Indication

Predominantly in cases of coccygodynia or S1 sciatica and when postsurgical symptoms take the form of a postdiscotomy syndrome or a postfusion syndrome.

Technique

The sacral canal is the continuation of the vertebral canal. It starts at the same level as the first sacral vertebra and ends between the sacrum and the coccyx. The fused transverse and spinous processes of the first four sacral vertebrae and their periosteum form its posterior border, and the periosteum of the five sacral vertebral bodies makes up its anterior border.

The articular processes of the fifth sacral vertebra form the sacral horn. The sacral hiatus, the exit point for the sacral canal, is located between the two sacral horns. These are easy to palpate on slim patients. The sacral hiatus is located at the caudal tip of an equilateral triangle drawn from a line connecting the two posterior superior iliac spines (PSIS). This aids orientation when searching for the insertion site, e. g., on obese patients (**Fig. 9.98**).

The patient is placed lying on one side or in a knee–elbow position. An 8–10 cm puncture needle is inserted through the connective tissue plate into the sacral canal. Aspiration is used to ensure that neither blood vessels nor the subarachnoid cavity, which is filled with cerebrospinal fluid, has been punctured.

Effect of the Sacral Epidural Injection

We use the sacral hiatus especially for access during epidural anesthesia (caudal anesthesia) in the treatment of postdiscotomy or postfusion syndromes. The sacral hiatus is the only access point available to reach the lumbar epidural space following fusion when the interlaminar access is blocked by bone grafts.

The disadvantage of the sacral technique is that a considerable volume of active substance has to be administered to ensure that the desired concentration reaches the affected nerve roots. Sacral epidural injections using a contrast agent and CT monitoring have demonstrated that the injected fluid is evenly distributed in the epidural

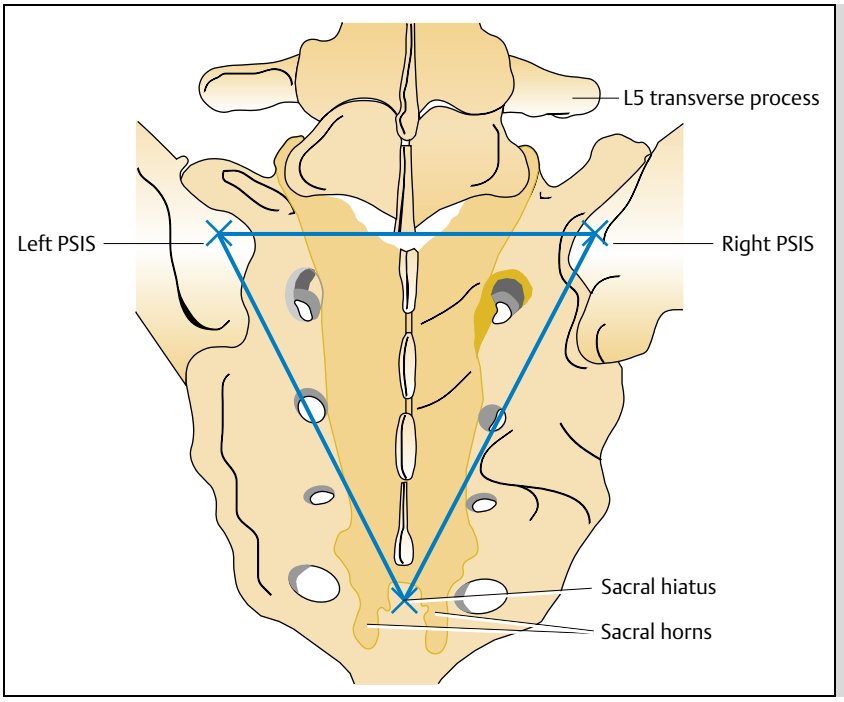


Fig. 9.98 Locating the sacral hiatus between the sacral horns.

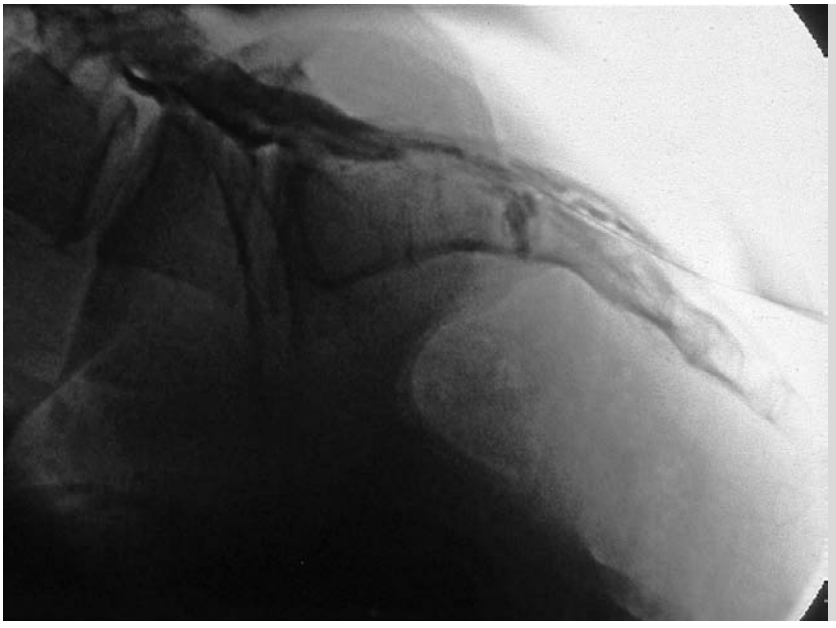


Fig. 9.99 Image-guided sacral epidural injection: The needle is inserted through the sacral hiatus into the peridural space in the sacral canal.

space and accumulates in large areas, particularly in the inferior part of the lumbar spine (**Fig. 9.99**).

A further disadvantage of the sacral technique is the position of the cannula deep in the anal cleft, with an associated increased risk of infection.

Sacral Epidural Injection Procedure (Figs. 9.100–9.116)

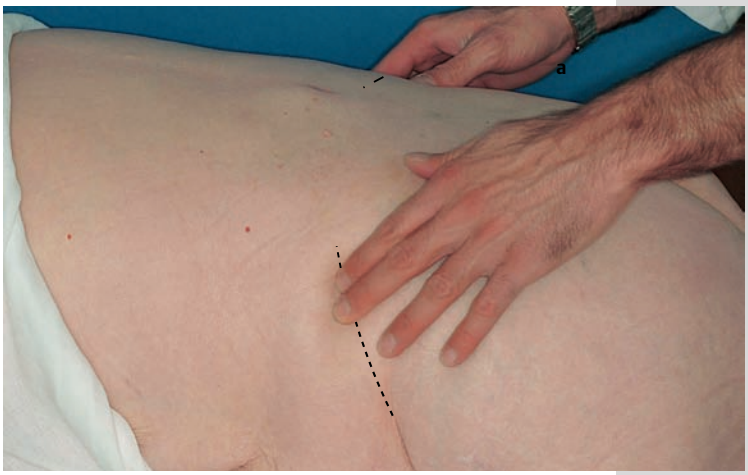
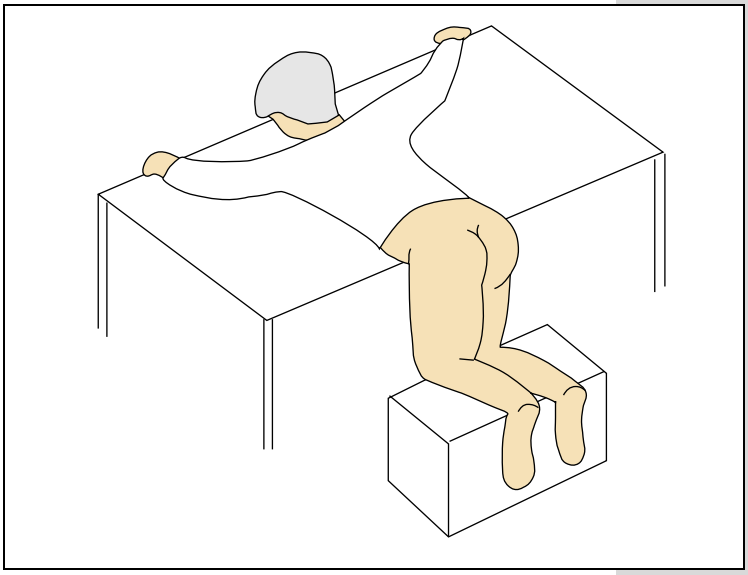


Fig. 9.100 a The examiner stands directly behind the patient, who kneels on a stool with the upper body resting on a higher examination couch. A pulse oximeter is attached. The examiner must be comfortably able to palpate the iliac crests and the border of the sacroiliac joints bimanually. The iliac crest, PSIS, and spinous process are always palpable in this position, even on obese patients.
b Schematic illustration of a patient kneeling with the upper body resting on the examination couch during a sacral epidural injection.



b

Fig. 9.101 Bimanual palpation of the medial sacral crest to both sacral horns.

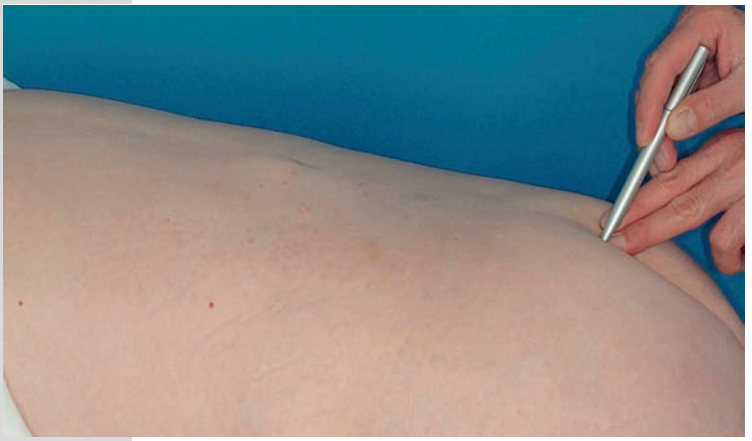


Fig. 9.102 Marking the injection site (the exit point of the sacral canal) using a pen with a retracted ink cartridge.

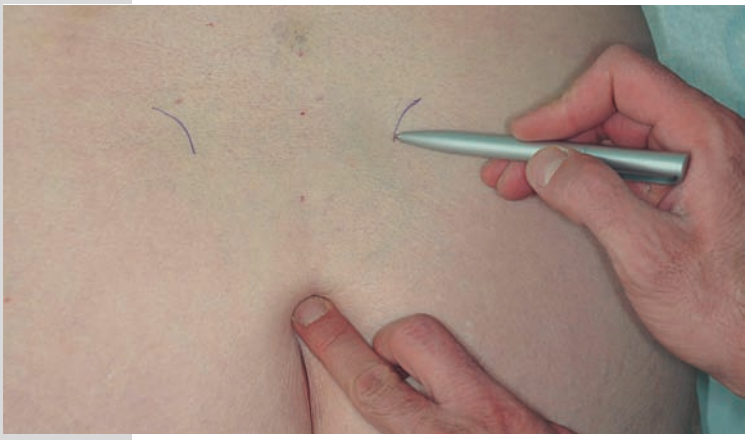


Fig. 9.103 Marking the iliac crests and the posterior superior iliac spine bilaterally.

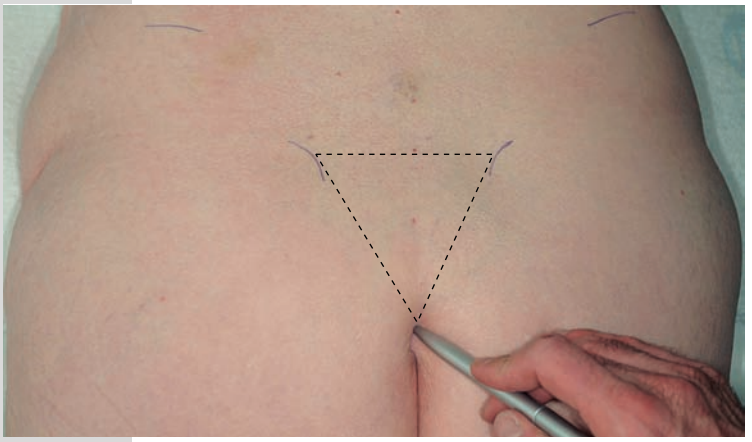


Fig. 9.104 Checking the position of the marked injection site. The sacral hiatus is located at the caudal tip of an equilateral triangle drawn from a line connecting the two posterior superior iliac spines (PSIS).

Fig. 9.105 The anal mucosa is protected with a swab placed in the cleft. Thorough disinfection (at least 3 min) follows.

Fig. 9.106 Sterile drawing up of the suspension containing 10 mL saline solution and, in some cases, 1–2 mL local anesthetic.

Fig. 9.107 The suspension is supplemented with 20–40 mg triamcinolone.

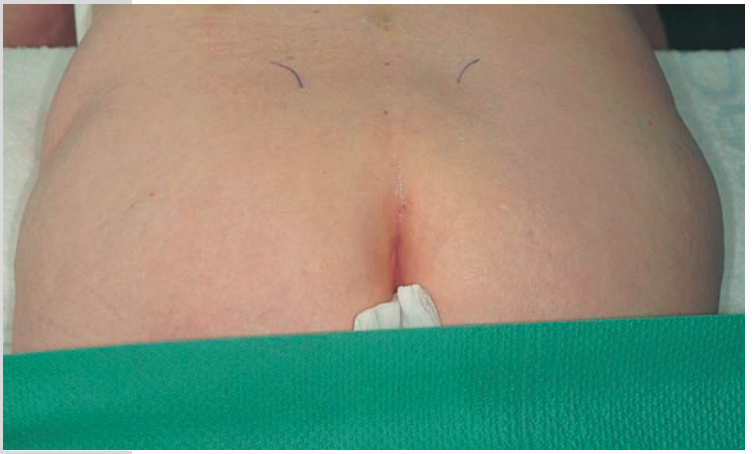


Fig. 9.108 Positioning a sterile towel.

Fig. 9.109 Disinfecting again (for at least 1 min more).

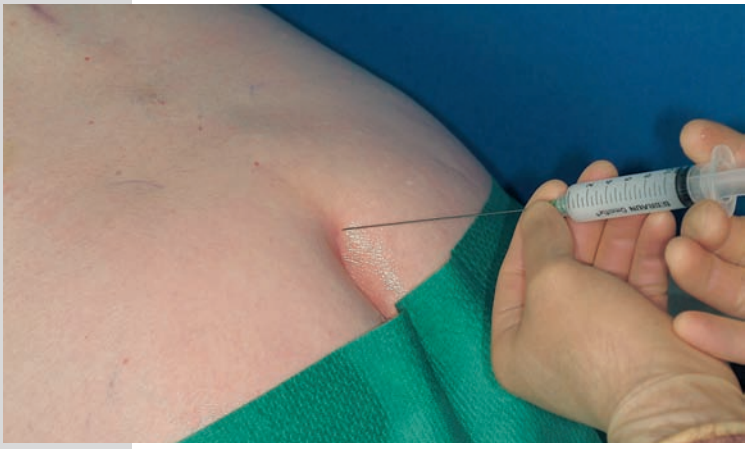


Fig. 9.110 The injection needle is inserted in the midline at the marked site just distal to the sacral hiatus at an angle of approximately 70° until bony contact is made. Both hands are used to guide the injection system. The left hand rests on the patient when necessary.
Caution: Subcutaneous administration of local anesthetic should be avoided as this makes palpation of the sacral horns more difficult.

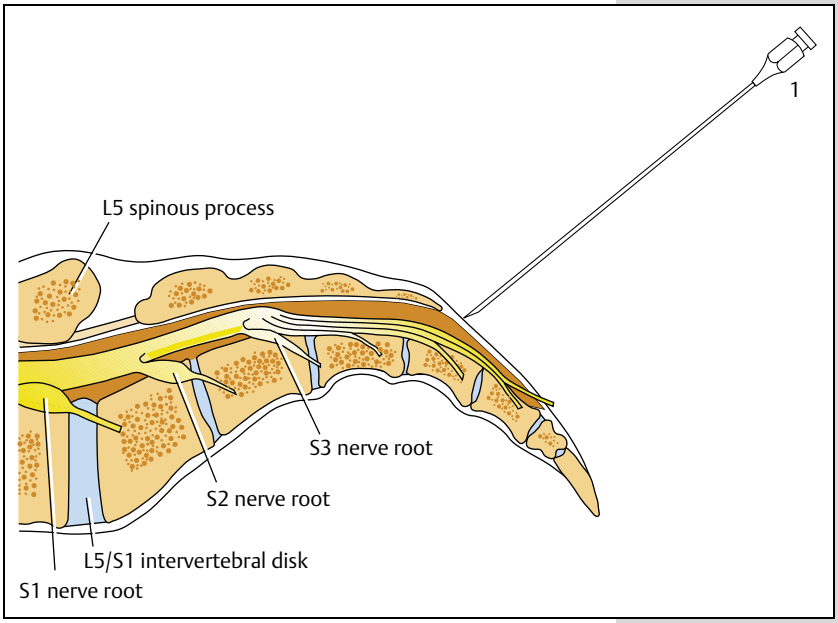


Fig. 9.111 Schematic illustration showing the needle position (1) before it is inserted into the sacral hiatus.

Fig. 9.112 Needle position, demonstrated on a skeleton.

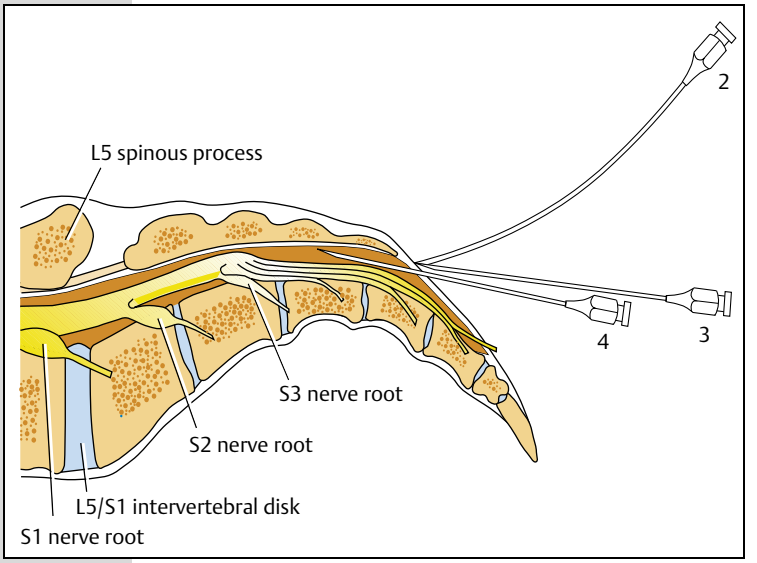


Fig. 9.113 Following this, the needle is retracted slightly, lowered caudally, and seeks out the sacral hiatus between the sacral horns (2). The resistance from the sacrococcygeal membrane is felt. The resistance is overcome—the cannula shaft now forms the continuation of the sacrum (3)—and the cannula is inserted ~3–4 cm further into the sacral canal under constant aspiration. (4) The tip of the cannula should not exceed the height of S2, or even better, S3.
Caution: Dural puncture.

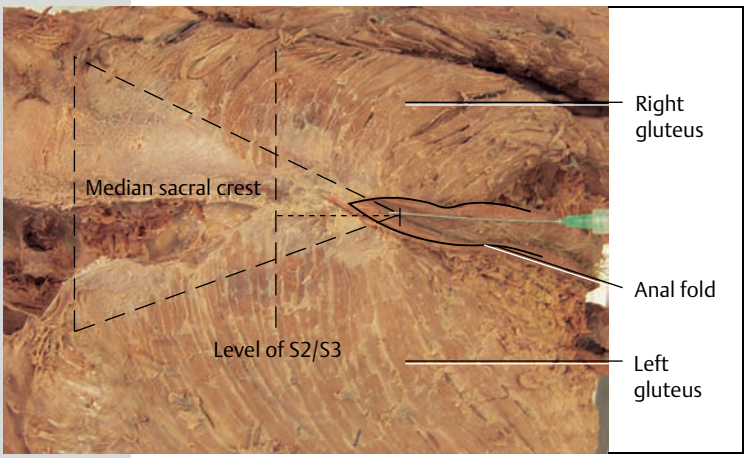


Fig. 9.114 Final needle position for the sacral epidural injection, demonstrated on an anatomical specimen. The needle is finally inserted only 3–4 cm.

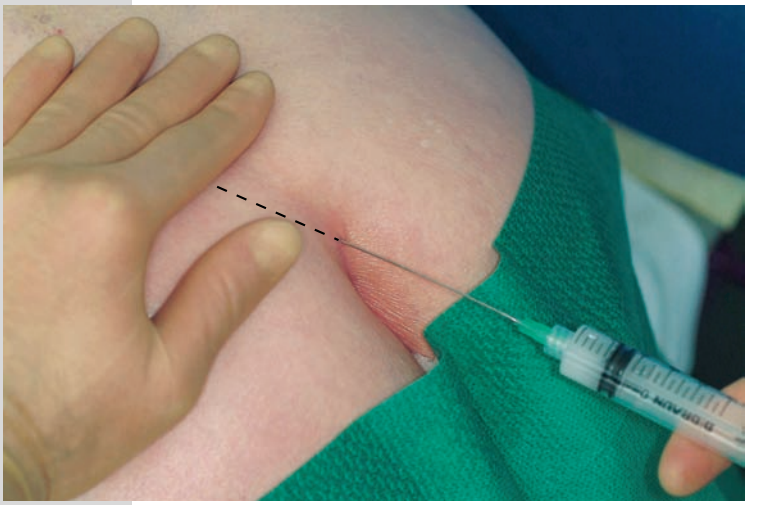


Fig. 9.115 Incorrect needle position (such as in the posterior soft tissue) is prevented by placing the other hand flat over the sacrum where it is assumed that the needle will end up and injecting a small amount of the suspension once an attempt has been made to aspirate. Slow injection is permitted when neither blood nor cerebrospinal fluid is seen coming out of the end of the cannula either spontaneously or following aspiration. Correct positioning enables the injection to be conducted without resistance. Patients often report pins and needles in the feet or a dragging pain in the thigh during the injection (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

Fig. 9.116 The needle is removed and a nonallergenic adhesive dressing, which should be removed after an hour, is placed over the injection site.

Posterior Epidural Injection (Epi-Posterior)

Principle

Injection via the interlaminar space into the posterior epidural space of the affected lumbar vertebral motor segment.

The interlaminar insertion of an injection cannula into the posterior epidural space of the lumbar vertebral canal is common in peridural anesthesia, and in orthopedic pain therapy when treating lumbar nerve root syndromes. Peridural anesthesia achieves complete analgesia by flooding primarily healthy nerve roots with a highly concentrated local anesthetic via a peridural catheter. In contrast, **the aim of orthopedic pain therapy** is to **flood compressed nerve roots** with an anti-inflammatory using **repeated individual injections**. When required, a dilute local anesthetic is added to reduce the sensitivity to pain.

Indication

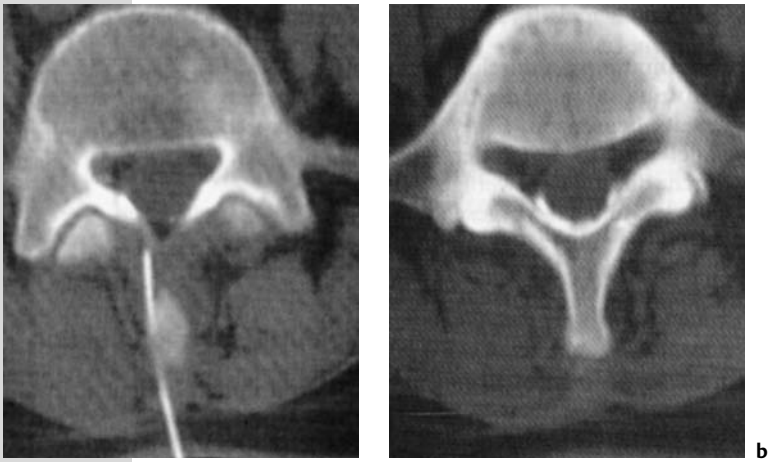
The posterior interlaminar injection technique reaches several nerve roots simultaneously. This can even occur bilaterally in some cases. This form of treatment is therefore mainly indicated in cases of central spinal canal stenoses and polyradicular syndromes.

Technique

Interlaminar access at L5/S1, L4/L5, or higher is selected, depending on which nerve root is affected. We usually select L3/L4 or L4/L5 in cases of spinal canal stenosis. Anteroposterior images of the lumbar spine should be available and displayed on a screen to visualize the interlaminar space of the respective segment and to assess whether the spaces are symmetrical. In the absence of an interlaminar gap, e. g., due to overlapping laminae, a neighboring segment that enables easier access is selected at the start. To achieve a targeted segmental/epidural injection, the procedure is identical to that for a lumbar puncture. The patient is placed in a sitting position. The mandrel needle (or the syringe needle system directly) is inserted between the spinous processes of the affected segment, through the ligamentum flavum, and into the peridural space. The dura should not be punctured. To avoid puncturing the dural sac, the mandrel is removed just before or during the perforation of the ligamentum flavum, a fluid-filled syringe is attached, and the needle is further inserted while pressure to the plunger is maintained, until the pressure on the injection suddenly subsides (loss of resistance). Once the position of the needle has been correctly established, a saline solution combined with a cortisone crystal suspension and, in some cases, local anesthetic, is injected.

The disadvantage of the posterior epidural injection technique is that a considerable volume of active substance has to be administered to ensure that the desired concentration reaches the affected nerve roots. The use of a contrast agent and CT monitoring of posterior epidural injections has demonstrated that the injected fluid is evenly distributed in the epidural space, with large areas of accumulation, particularly in the posterior section (**Fig. 9.117a**, **b**).

Fig. 9.117a, b Posterior epidural injection. The contrast agent accumulates mainly in the posterior epidural space.



a b

Posterior Epidural Injection Procedure (Figs. 9.118–9.141)

Fig. 9.118 An anteroposterior image of the inferior lumbar spine should always be in the physician's visual field when conducting the posterior epidural injection with medial interlaminar access and the loss-of-resistance technique. Note the interlaminar space in the segment to be injected into.

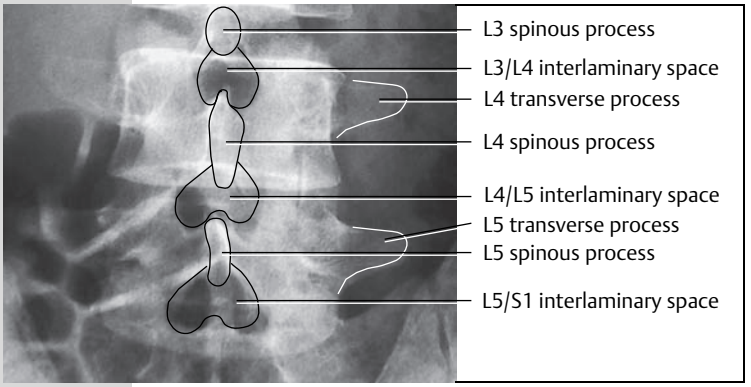
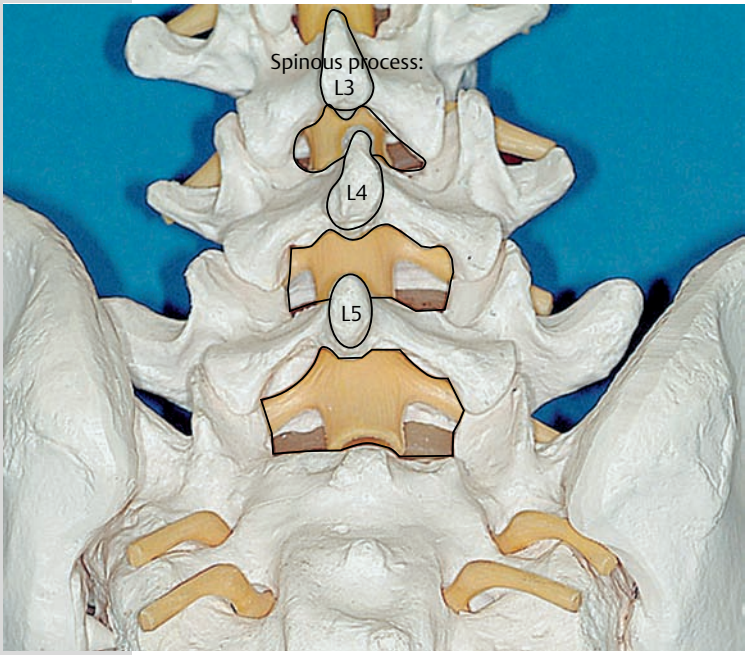


Fig. 9.119 The interlaminar spaces and spinous processes, demonstrated on a skeleton. The interlaminar space widens up particularly when the spine is slightly flexed, enabling easy access into the vertebral canal.



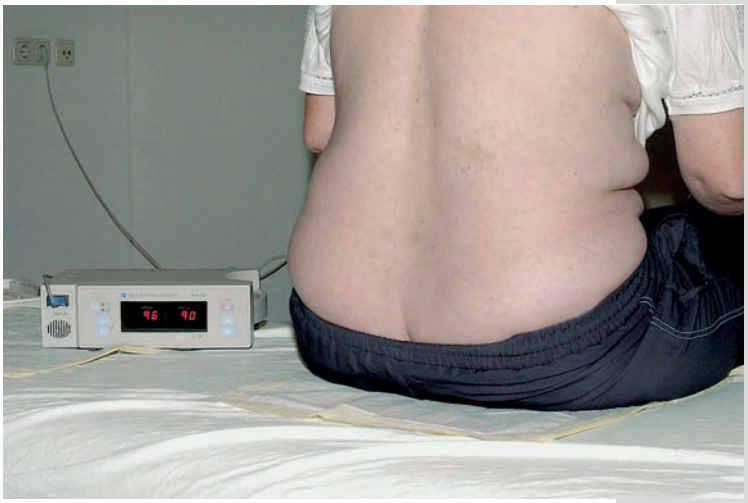


Fig. 9.120 Patient position from posterior for the posterior epidural injection. A pulse oximeter is located next to the patient.

Fig. 9.121 Arrangement of patient, physician, and assistant. The patient should be sitting on a high examination couch so that the sitting physician can comfortably palpate the iliac crests (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

Fig. 9.122 Bimanual palpation and orientation, demonstrated on a skeleton.

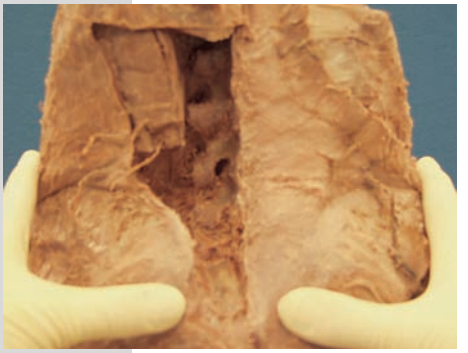


Fig. 9.123 Bimanual palpation of the bilateral posterior superior iliac spines and iliac crests, demonstrated on an anatomical specimen.

Fig. 9.124 Bimanual palpation and orientation on the sitting patient. The thumbs slide medially from a lateral position, over the posterior iliac spine, and in the sulcus between the iliac spine and the medial sacral crest. Index and middle fingers palpate the iliac crest.

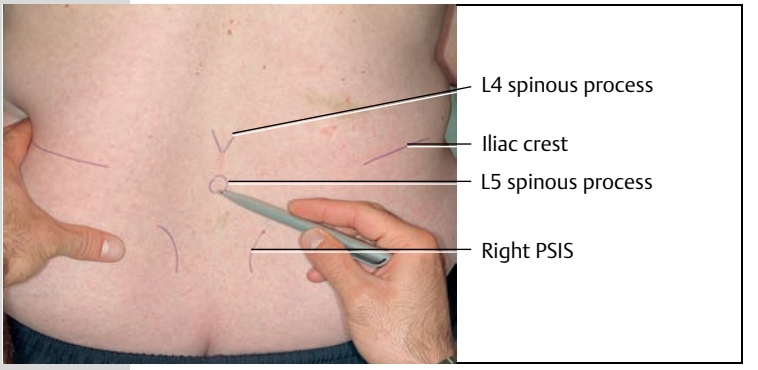


Fig. 9.125 Marking the tips of the L4 and L5 spinous processes in relation to the iliac crest. The connecting line between the iliac crests corresponds to the level of the L4 spinous process.

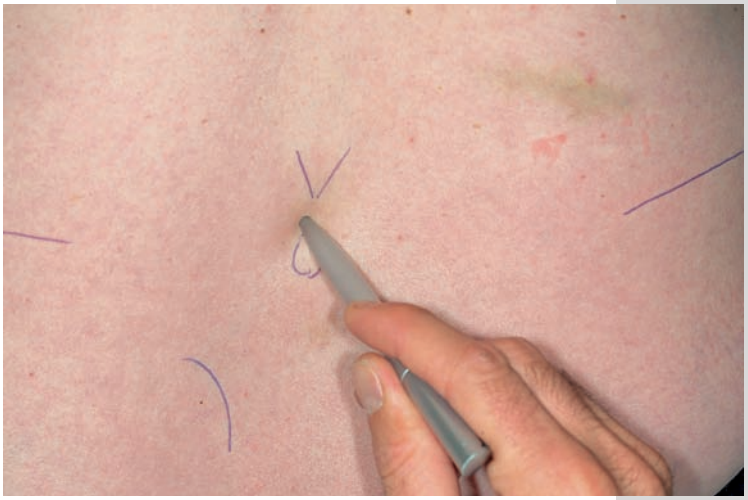


Fig. 9.126 Marking the injection site with a pen (with retracted ink cartridge) exactly in the midline between the L4 and L5 spinous processes.

Fig. 9.127 The pen indicates the marked point between L4 and L5 on a skeleton.

Fig. 9.128 Towels are placed to protect clothing. The disinfection process can then begin (at least 3 min). Ink marks may be removed again during the skin disinfection but the pressure-marked site, required for the posterior epidural injection at the L4/L5 segment, remains visible.

Fig. 9.129 The patient sits in a slightly flexed posture for the injection, with their feet on a chair. Sterile gloves and mask are necessary for several reasons, including verbal monitoring when the cannula is being manipulated (e. g., when connecting the end of the syringe).

Fig. 9.130 Sterile drawing up of 10 mL fluid (e. g., saline solution) into a syringe, as well as a 10 mL syringe with saline and cortisone suspension (not illustrated here).

Fig. 9.131 It is advisable to use a relatively large cannula (e. g., Spinocan 21 G). Thinner cannulas should not be used under pressure as it is necessary to feel the sudden loss of resistance when the tip of the needle has reached an epidural position.

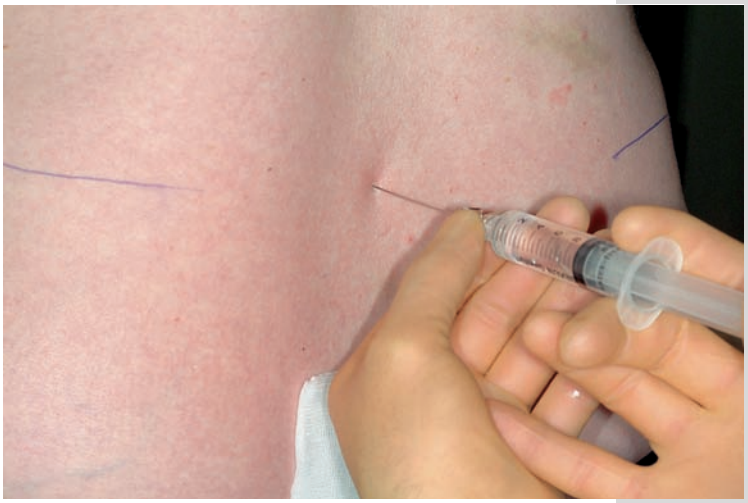


Fig. 9.132 Slowly inserting the cannula with the attached saline syringe under pressure (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

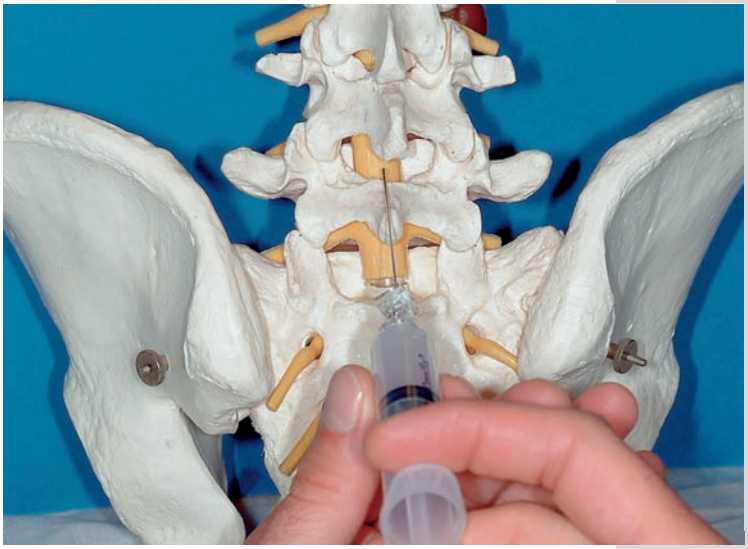


Fig. 9.133 Position of the needle for the posterior epidural injection between L4 and L5, shown on a skeleton.

Fig. 9.134 The syringe is removed after a sudden loss of resistance is felt when inserting the needle with the saline syringe. This is necessary in order to check for drops of cerebrospinal fluid (ask the patient to cough).

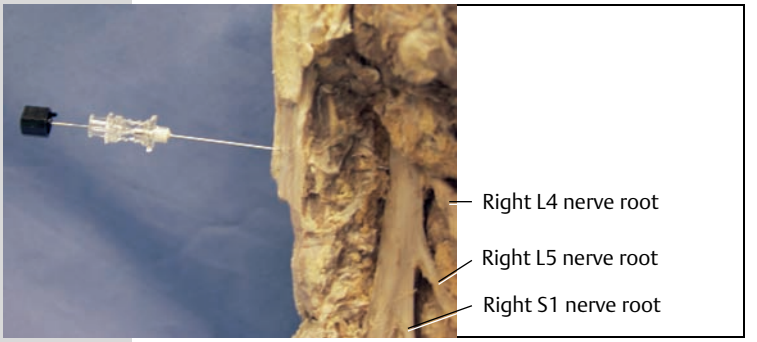


Fig. 9.135 Position of the needle during a posterior epidural injection, demonstrated on an anatomical specimen (right lateral/posterior view, right laminae and soft tissue from L4 to S1 have been dissected). The cannula passes through the skin, interspinous ligament, fatty tissue between the spinous processes, and the ligamentum flavum. It is only after passing through these tissues that the loss of resistance is felt.

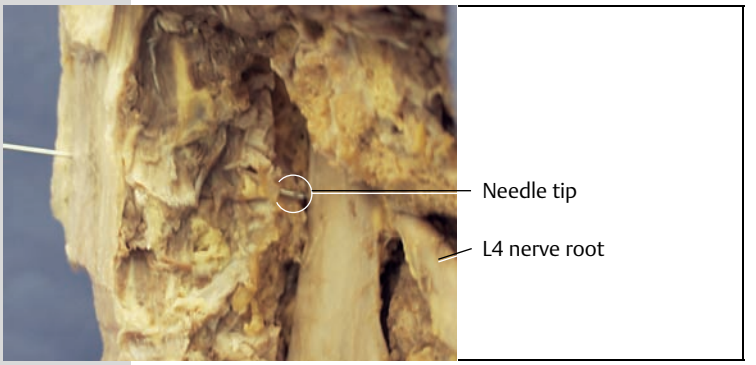


Fig. 9.136 Needle position during the posterior epidural injection. The tip of the needle is found in the epidural space, behind the dura.

Fig. 9.137 Exchanging the saline syringe for the cortisone–saline syringe.

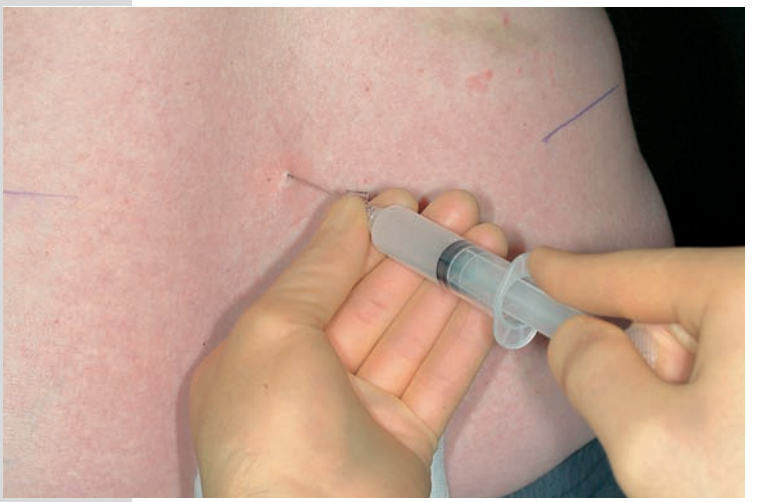


Fig. 9.138 Attempt to aspirate before injecting.

Fig. 9.139 Slowly injecting the cortisone–saline solution into the posterior epidural space. Correct positioning allows the injection to be conducted without resistance (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

Fig. 9.140 The needle is removed and a nonallergenic adhesive dressing is placed over the injection site. The dressing should be removed after an hour.

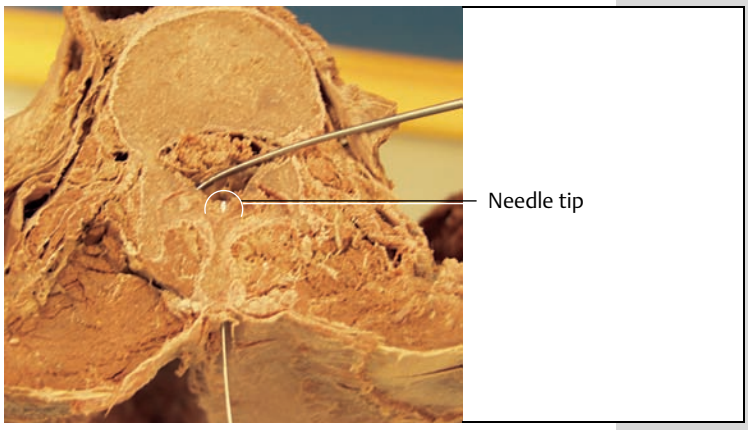


Fig. 9.141 Overview of the needle position during the posterior epidural injection. The dissector has pushed the dura and the cauda equina fibers in an anterior direction. The tip of the needle is found in the posterior epidural space.

Epidural Perineural Injection (Epi-Peri)

Principle

Injection of small amounts of steroids and local anesthetics into the anterolateral epidural space using an oblique interlaminar access and the double-needle technique.

Indication

Monoradicular lumbar nerve root irritation due to displaced intervertebral disk tissue and/or bony compression in cases of lateral spinal canal stenosis. The technique also enables targeted periradicular infiltration in cases of nerve root irritation caused by postsurgical scarring (postdiscotomy syndrome).

Technique

The injection is administered with the patient in a sitting position. An introducer needle is inserted **1 cm inferior** and **1 cm contralateral** to the L5 spinous process at an angle of 10–20° until it reaches the ligamentum flavum or, in some cases, just before this point. A **12 cm 29 G cannula*** is inserted into the introducer needle until the needle tip is felt to make contact with the bone. If bone (lamina) is contacted too early, the injection angle must be modified, depending on the situation, along the frontal or sagittal plane. As in the posterior injection technique, it is advisable to have an anteroposterior radiograph of the lumbar spine available, to assess the extent of the interlaminar space. About 20 % of patients report a slight radiating pain, but this is kept within reasonable limits when the thin 29 G cannula is used. A total of 1 mL of local anesthetic (ropivacaine 2 mg/mL, levobupivacaine 2.5 mg/mL, or 5 mg triamcinolone) is injected. This injection can be carried out under CT monitoring for training purposes (**learning curve**) and for scientific documentation.

Effect of the Epidural Perineural Injection

The administration of fluids into the anterolateral epidural space reaches the spinal nerve roots in the lateral recess as they exit the dural sac in the L4/5 and L5/S1 segments. In addition, the local anesthetic reaches the spinal ganglion by entering the intervertebral foramen. Small amounts of dilute local anesthetic usually achieve a reduction in back and leg pain without affecting motor function. Temporary signs of paralysis or feelings of lameness in the relevant area are to be expected in less than 5 % of all cases. Patients must be made aware of this in advance and precautions must be take accordingly.

NOTE

The aim of **epidural perineural injections** is not the complete analgesia and paralysis of the spinal nerves found in the epidural space, as is the case when preparing for surgery. Rather, they aim to reduce pain and desensitize irritated neural structures in the lumbar vertebral motor segment **directly at the origin of the pain**.

Epidural Perineural Injection Procedure (Figs. 9.142–9.172).

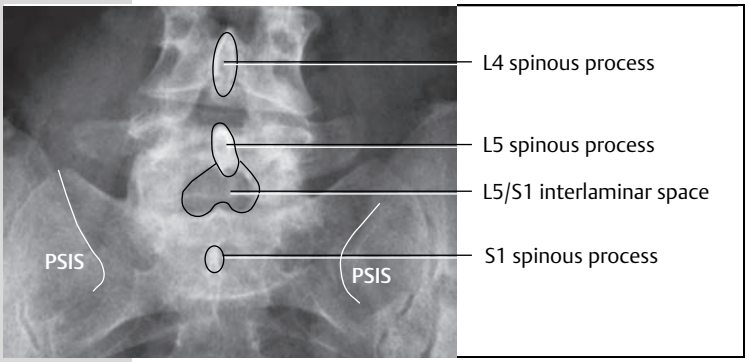


Fig. 9.142 An anteroposterior image of the inferior lumbar spine should be within the physician's visual field when conducting the epidural perineural injection with oblique interlaminar access and the double-needle technique. The standard interlaminar space to be sought out for lumboischialgia, i. e., for a L5 and/or S1 nerve root compression, lies between the L5 arch and the upper border of the sacrum. This interlaminar space is also easily visible in the presence of scolioses, osteoporosis, and all types of deformations. It allows a suitably long needle (at least 12 cm) to pass through it when using the palpatory anatomical orientation points.

* Available at B. Braun Medical Inc., USA.

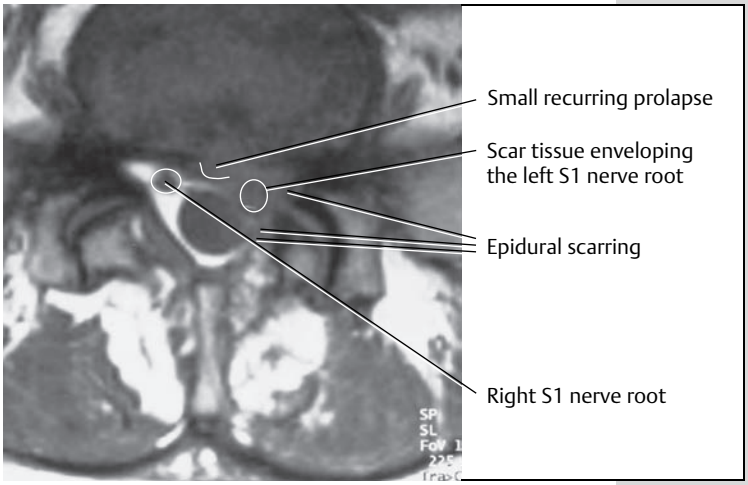


Fig. 9.143 MRI of the lumbar spine: Transverse slices at the level of L5/S1, postdiscotomy syndrome with small recurring prolapse. Signs of recurring clinical nerve root irritation at L5 and S1 on the left.

Fig. 9.144 Arrangement of patient, physician, and assistant: The patient sits on a high examination couch with their feet on a stool. This ensures that a large amount of trunk flexion is possible, with the associated widening of the interlaminar access points. The assistant verbally monitors the patient and maintains body contact. The physician palpates, sitting on a low stool. The iliac crests and the spinous processes are at the physician's eye level (from Theodoridis T, Ludwig J, Krämer J. Injektionstherapie an der Lendenwirbelsäule. In: Jerosch J, Steinleitner W. *Minimal-invasive Wirbelsäulen-Intervention*. Cologne, Germany: Deutscher Ärzte-Verlag; 2005).

Fig. 9.145 Epidural perineural injection, here at the left L5/S1 segment, and its typical indication: Postdiscotomy syndrome following a left L5/S1 intervertebral disk operation. The areas to be palpated should be free of clothing. The skin must not display signs of infection or other disease.

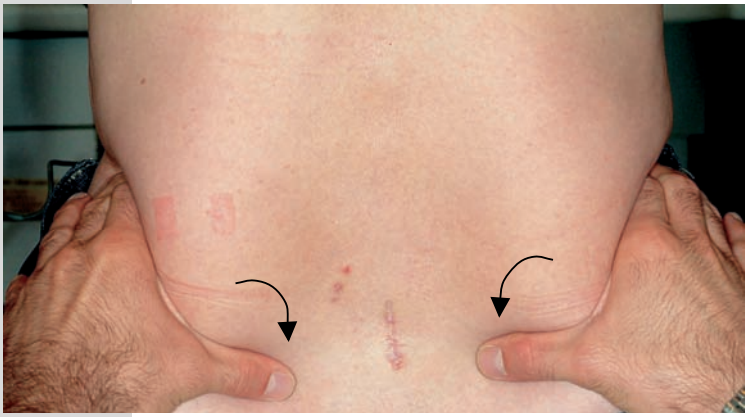


Fig. 9.146 Palpation and orientation, demonstrated on a patient: The thumbs slide medially from a lateral position, over the posterior iliac spine, and in the sulcus between the iliac spine and the medial sacral crest. Index and middle fingers palpate the iliac crest. The L4 spinous process is found at the same level as the transverse line connecting the two upper iliac crests. The L5 spinous process is found below this. The injection site is found directly below the L5 spinous process.

Fig. 9.147 Bimanual palpation, demonstrated on a skeleton.

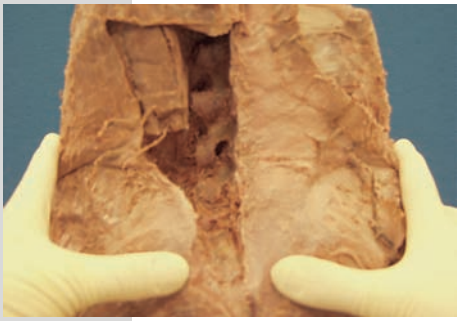


Fig. 9.148 Bimanual palpation, demonstrated on an anatomical specimen. The superior iliac spine and the posterior superior iliac spine orientation points that need to be palpated are not covered by muscles but only surrounded by relatively soft, subcutaneous, fatty tissue, so that it is possible to palpate the prominent bones deeply even on obese patients.

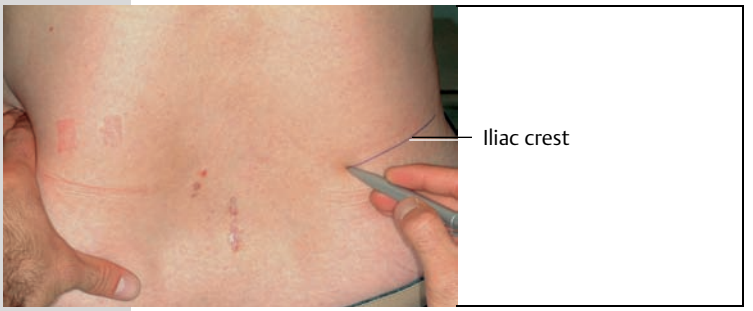


Fig. 9.149 Transverse connecting line at the same level as the iliac crests. The L4 spinous process is found at this height.

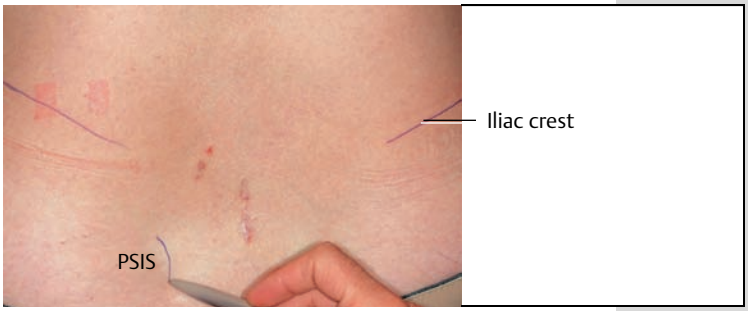


Fig. 9.150 Marking the posterior superior iliac spine (PSIS).

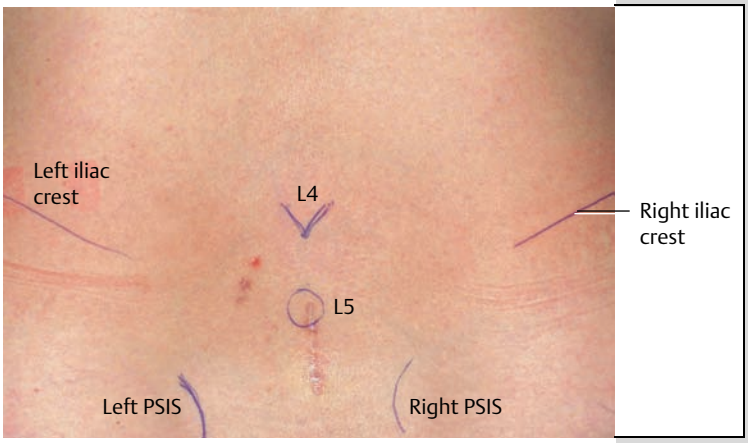
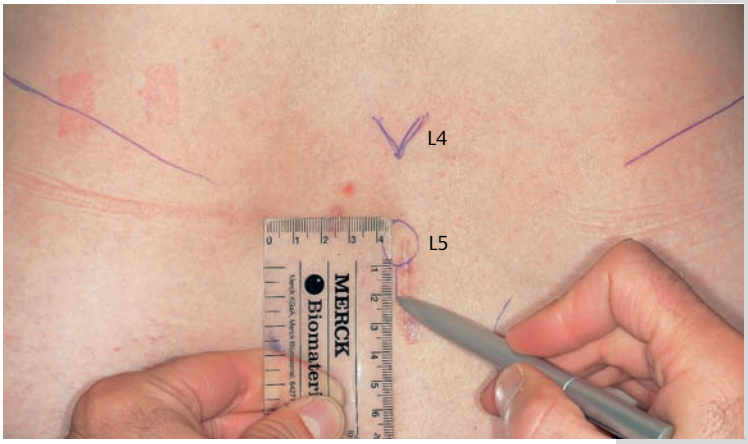
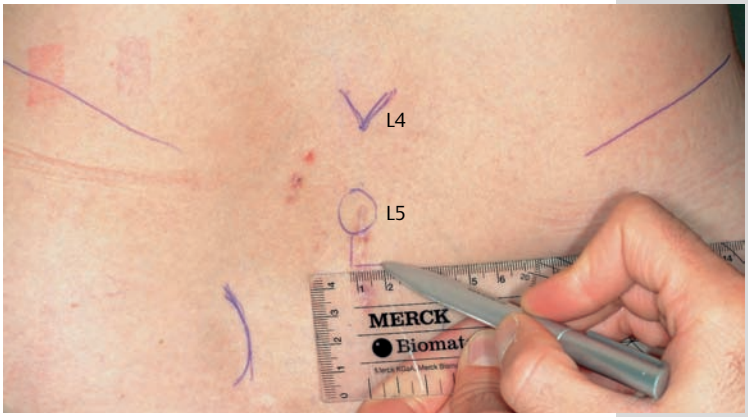


Fig. 9.151 Marking the L4 and L5 spinous processes. The scar can be observed directly beneath (L5).



a



b

Fig. 9.152a, b The insertion site is marked 1 cm inferior to the palpable tip of the L5 spinous process (**a**) and 1 cm contralateral, on the opposite side of the affected nerve root (**b**).

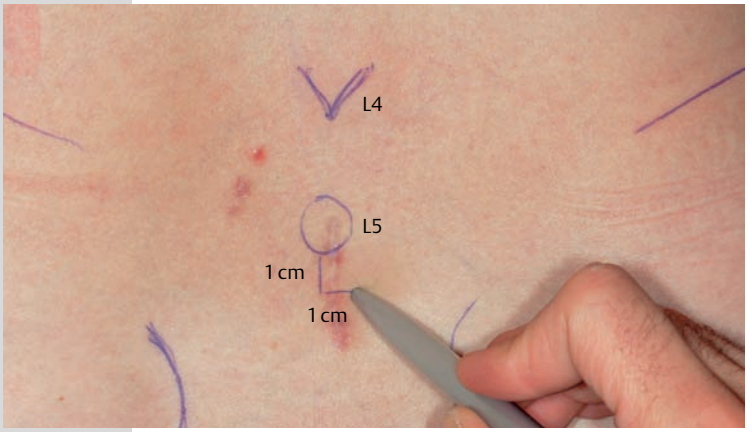


Fig. 9.153 Marking the injection site using a pen with a retracted ink cartridge.

Fig. 9.154 Towels are used to protect the patient's clothing. The skin is disinfected for at least 3 min.

Fig. 9.155 Sterile drawing-up of medication; package opened with sterile gloves. The cannulas (introducer cannula, 29 G cannula) are shaken out onto a sterile surface. An empty 1 mL aspiration syringe, a 1 mL syringe with 1 mL of local anesthetic needed to anesthetize the nerve root, and a 1 mL syringe that contains the agent to be injected into the epidural space (usually a cortisone crystal suspension).

Fig. 9.156 The physician sprays the injection site again for 3 min. By now the physician is wearing a mask, as he/she is constantly talking to the patient (verbal monitoring). The mask is necessary because of the needle exchange and manipulation between the introducer cannula and the actual injection cannulas, with the needle being inserted and retracted when the needle position is being corrected.

Fig. 9.157 The sterile phase (sterile gloves, mask) starts with the insertion of the introducer cannula 1 cm superior and 1 cm contralateral at the marked point and at a 15–20° angle until the ligamentum flavum is reached. The introducer cannula is so short that is can be fully inserted into normal patients, and especially into obese patients. For slim patients it should be inserted only halfway, to avoid puncturing the dural sac. If drops of cerebrospinal fluid enter the introducer cannula, retract the cannula a short way and then continue the injection procedure. The appropriate post-injection measures such as bed rest, the administration of fluids, etc. must be observed.

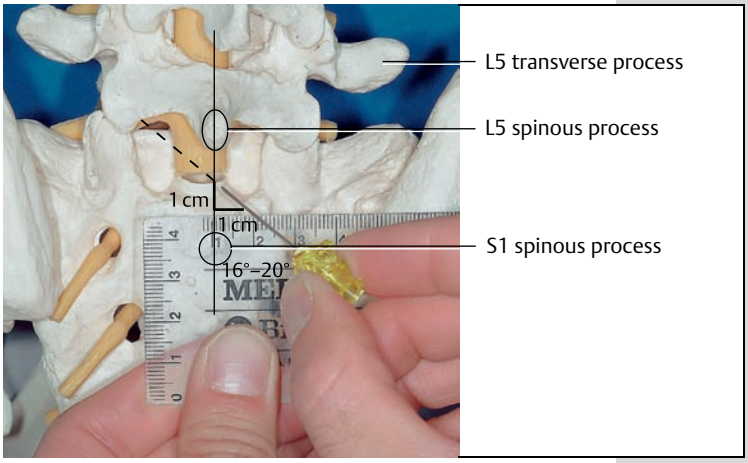


Fig. 9.158 The position of the introducer cannula on a skeleton with imaginary insertion 1 cm inferior and 1 cm contralateral to the tip of the spinous process. The needle passes through the interspinous ligament at the midline.

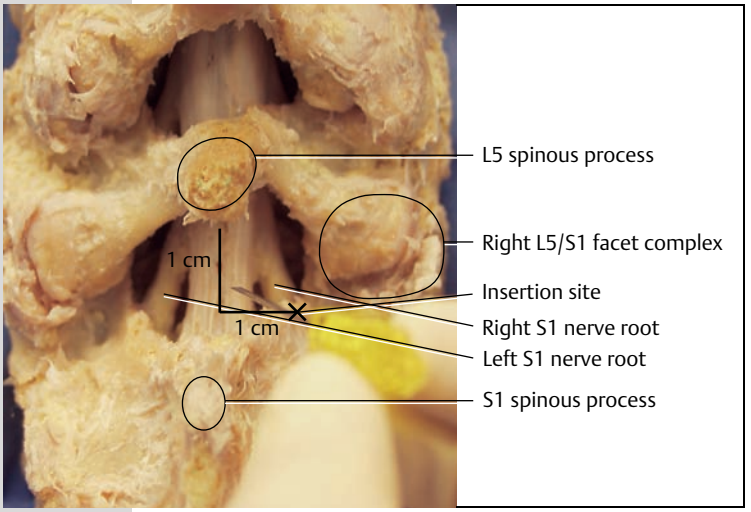


Fig. 9.159 The position of the introducer cannula, demonstrated on an anatomical specimen. The needle tip is also considerably posterior to the dural sac at the midline.

Fig. 9.160 Two-handed insertion of the introducer cannula to sound out the interlaminar space when early bony contact (vertebral arch) is made and the insertion direction of the needle is changed in a superior/inferior direction. This applies only to slim patients. Immediate insertion without bony contact applies for obese patients.

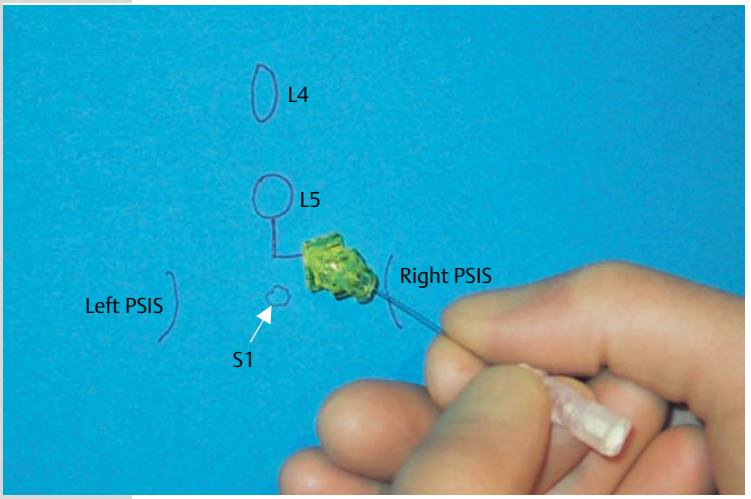


Fig. 9.161 Inserting the 29 G cannula into the introducer cannula. Do not touch the front part of the needle, even when wearing sterile gloves, as this part of the needle enters the vertebral canal. When resistance is felt in the deeper tissue and the needle bends at the rear end, it is possible—and in most cases necessary—to stabilize the rear end of the needle with the other hand. The needle is therefore inserted bimanually from this point onward.

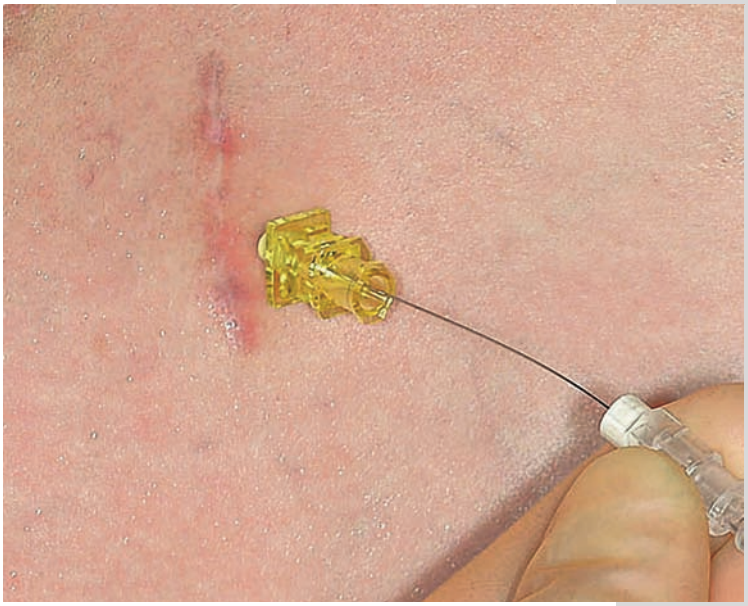


Fig. 9.162 Final position of the double-needle system, demonstrated on a patient.

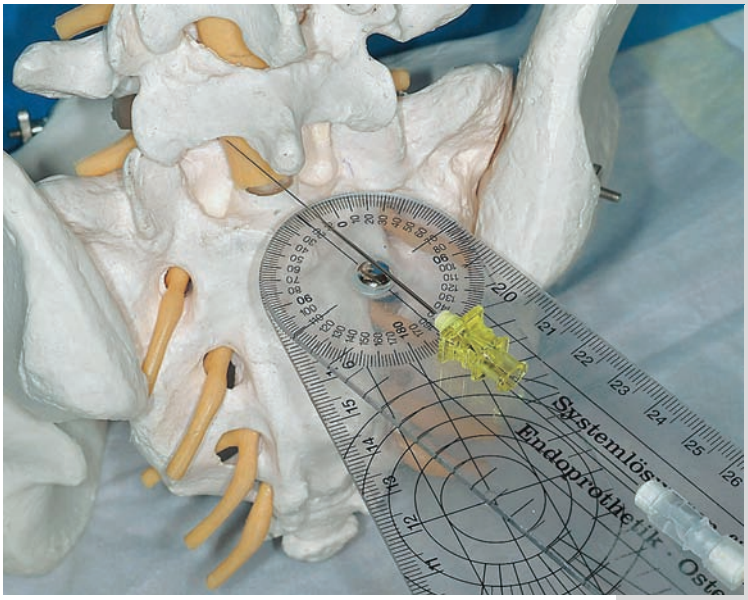


Fig. 9.163 Double-needle system position, demonstrated on a skeleton. The puncture channel deviates approximately 15–20° from the sagittal midline.

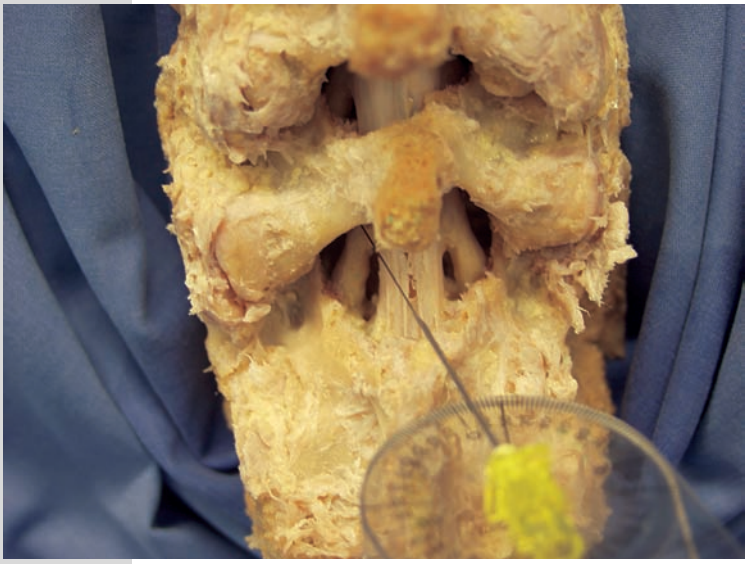


Fig. 9.164 Double-needle system position, demonstrated on an anatomical specimen. The lateral epidural space is relatively large at L5/S1 and has its maximum area at 15–20° between the lateral dural border and the medial bony border.

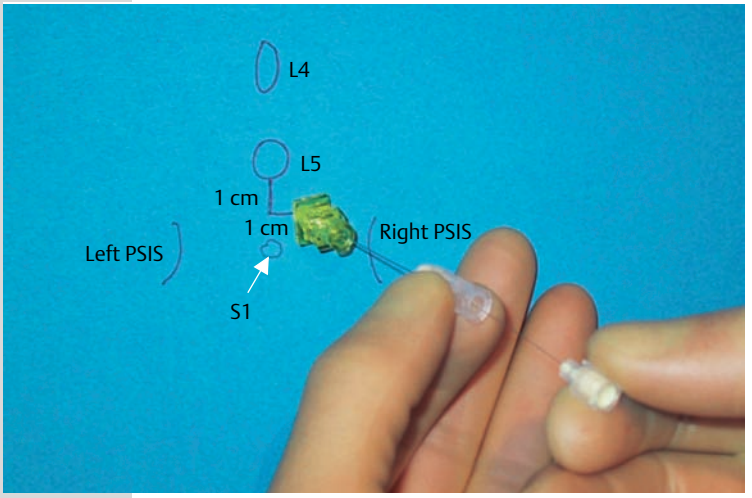


Fig. 9.165 Removing the mandrin from the 29 G cannula.

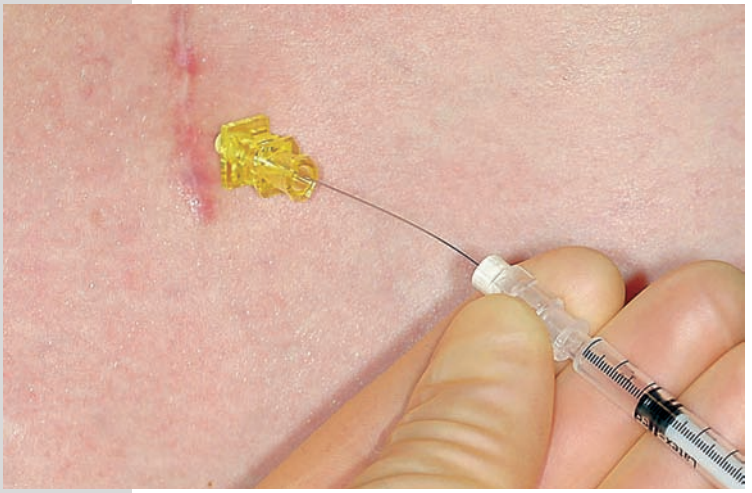


Fig. 9.166 Anterolateral epidural space, **Phase 1:** The 12 cm 29 G cannula is inserted until bony contact is registered. Aspiration using an empty insulin syringe. The aspiration of cerebrospinal fluid is unlikely at bony contact, but if it does occur, the needle tip in the epidural space must be corrected by inserting the needle again. Injection of 1 mL local anesthetic.

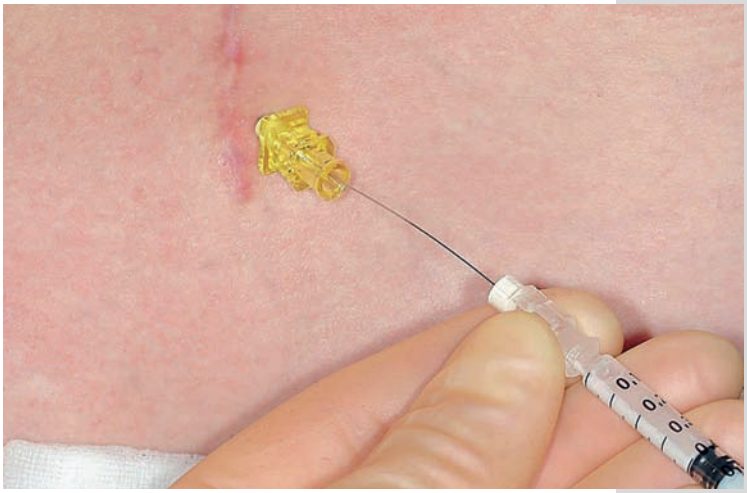


Fig. 9.167 Anterolateral epidural space, **Phase 2:** The anesthesia syringe is detached and a further insulin syringe is attached, filled with 1 mL of anti-inflammatory (usually in the form of a cortisone crystal suspension). Slow injection without encountering resistance.

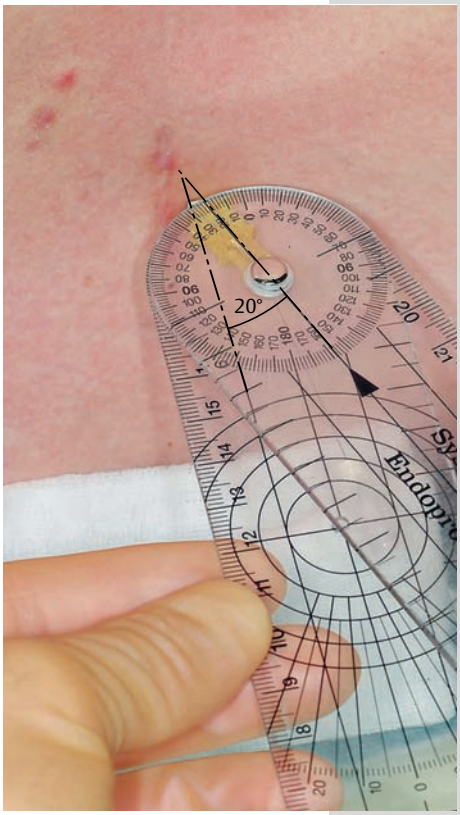


Fig. 9.168 Before removing the needle, the angle of the needle in the horizontal plane is measured and documented. This is normally between 15° and 20° but may be less than 10° when the facets are situated very close to each other, e. g., in cases of spinal canal stenosis.

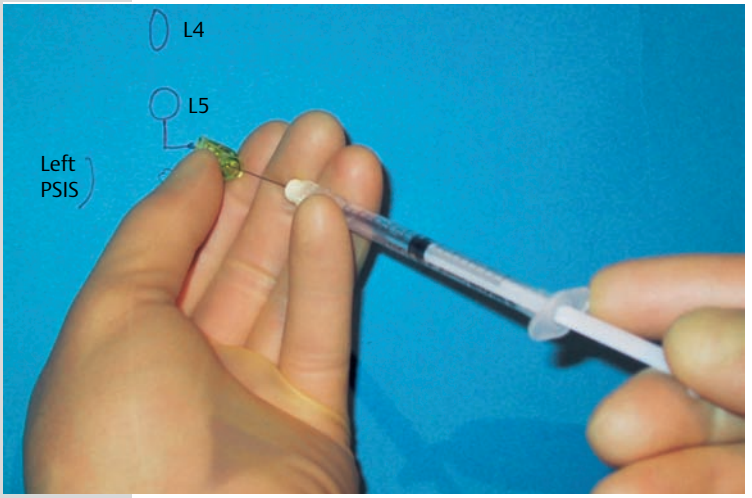


Fig. 9.169 Removing the needle system. Both needles are retracted **simultaneously** under constant aspiration. The first indication of possible penetration of the dural sac is the aspiration of cerebrospinal fluid when the needle is being retracted. This is expected in fewer than 10 % of all cases and should be documented in the medical record. The follow-up examination of several thousand patients has shown that this situation is not a complication and is not clinically significant, as a 29 G cannula has been used. The patient should nevertheless be informed that the thin needle had to be pushed through the dural sac because of the patient's particular anatomical proportions, and that this was necessary to reach the irritated nerve at the best possible location.

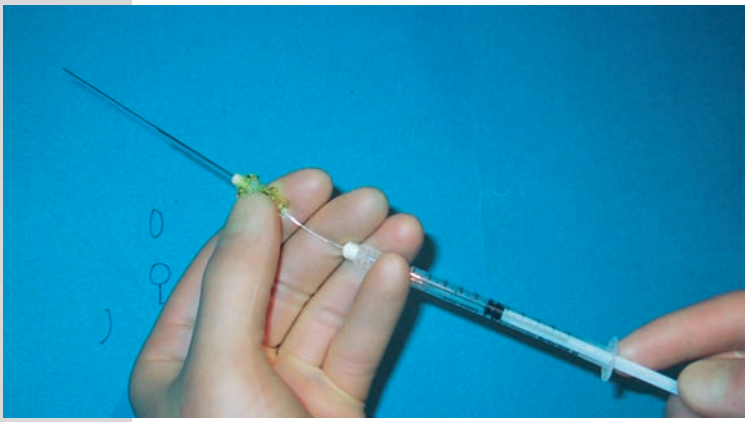


Fig. 9.170 The distance between the introducer cannula tip and the 29 G cannula tip remains constant when retracting both needles simultaneously. The entire needle length can be kept constant by bending the 29 G cannula just above the rear end of the introducer cannula.

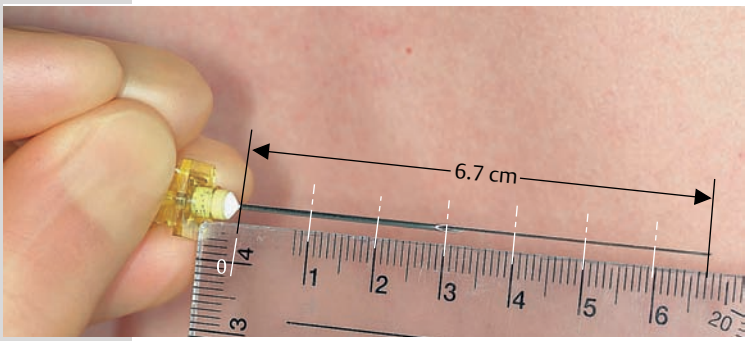


Fig. 9.171 The inserted needle length is measured after it has been withdrawn. This method is used to determine the distance between the skin and the bony contact in the anterolateral epidural space (6.7 cm here, as documented in the medical records).

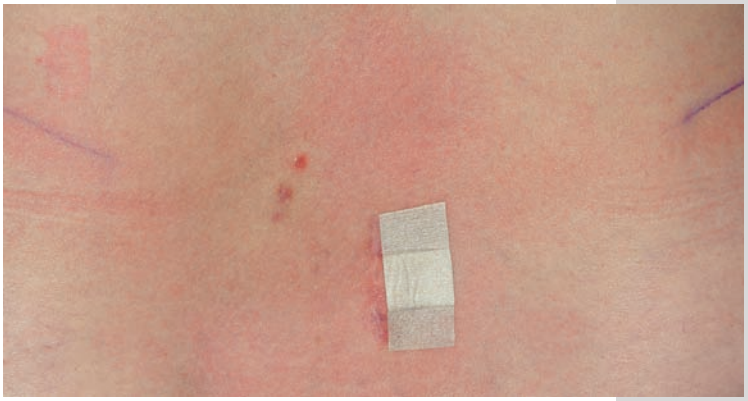


Fig. 9.172 Nonallergenic adhesive dressing that should be removed after one hour.

Epidural Perineural Injection (2nd Example)
(Figs. 9.173–9.182)

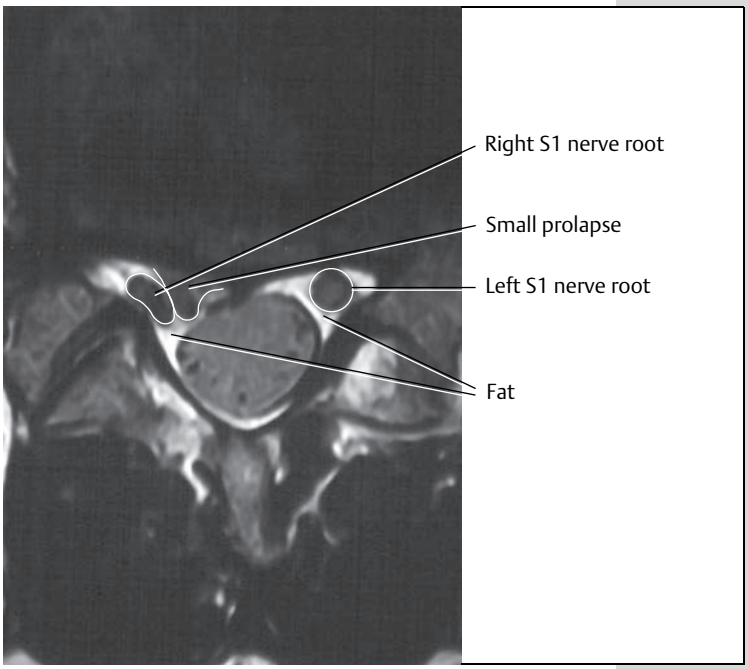


Fig. 9.173 Relatively small infradiscal prolapse applying pressure to the right S1 nerve root. The right S1 nerve root appears flattened in comparison to the nerve root on the opposite side. The width of the vertebral canal is normal and the epidural space is filled with a sufficient amount of fat (light-colored). There is good chance of success using nonsurgical interventions, such as the epidural perineural injection, by reducing the swelling in the nerve root and shrinking the size of the prolapse.

Fig. 9.174 The introducer cannula has passed through the interspinous ligament in the midline. The 29 G cannula is inserted at a 20° angle.

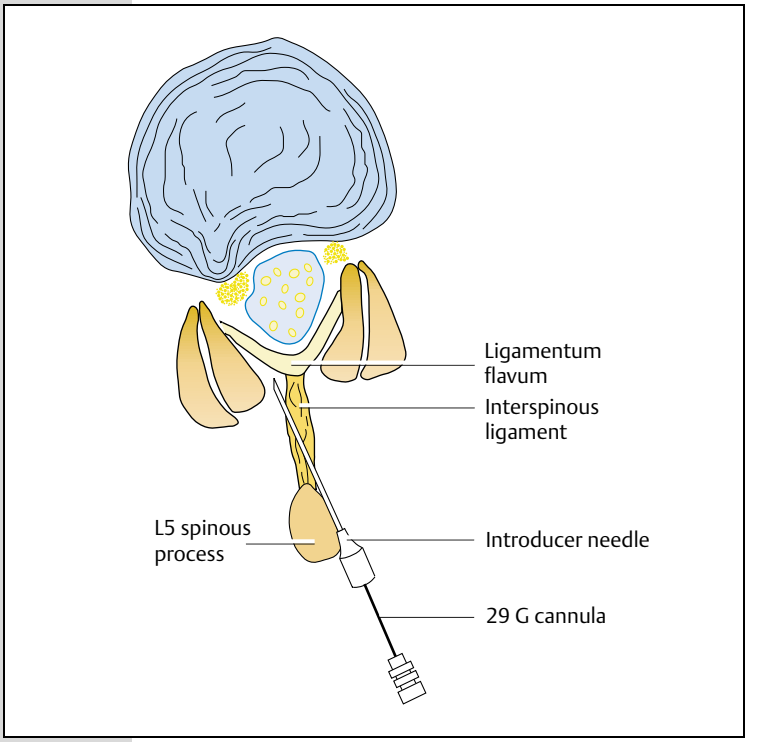
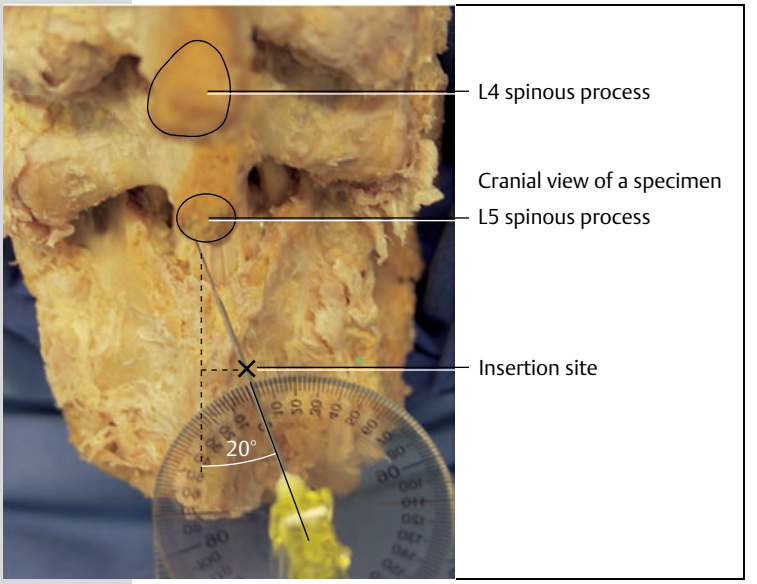


Fig. 9.175 Position of the introducer cannula, 1 cm inferior and 1 cm contralateral to the L5 spinous process at an angle of approx. 20°.



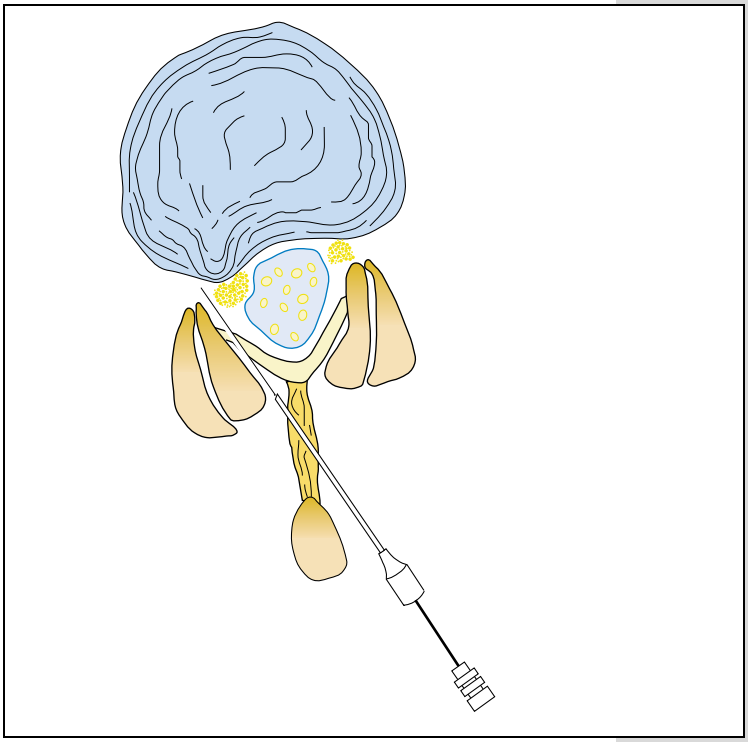


Fig. 9.176 Inserting the long 29 G cannula in the puncture channel between the medial section of the facet and the lateral section of the dural sac until contact is made with bone or the intervertebral disk in the lateral section of the anterior epidural space.

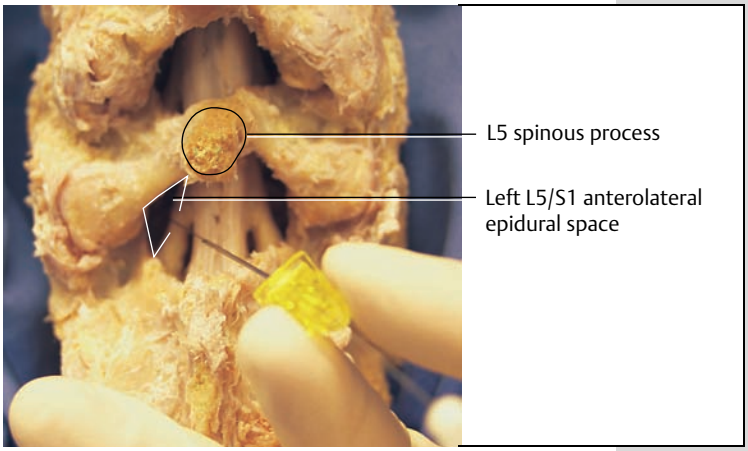


Fig. 9.177 The position of the introducer cannula is directed toward the anterolateral epidural space, demonstrated here on an anatomical specimen.

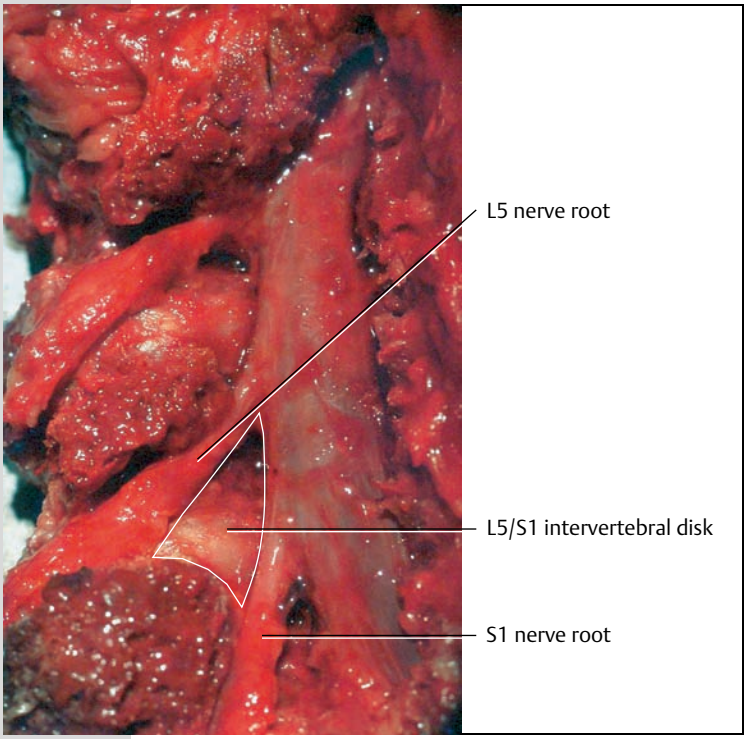


Fig. 9.178 Lumbar spine specimen after removal of the laminae and the facets, from a left lateral/posterior view. The L5 and S1 nerve roots and the superior edge of the sacrum form a triangle. The intervertebral disk is located in the inferior half of the triangle and the posterior surface of the L5 vertebral body is found in the superior half. The L5 nerve root passes through the L5/S1 intervertebral foramen between the L5 pedicle and the sacrum. The S1 nerve root traverses the posterolateral section of the L5/S1 intervertebral disk. The L5 nerve root initially lies in the lateral section of the intervertebral foramen at the same level as the intervertebral disk. **The triangle created by the L5 nerve root, the S1 nerve root, and the superior edge of the sacral base represents the "trouble spot" for lumboischialgia at L5 and S1.** The injection of an anesthetic and an anti-inflammatory reaches both the **L5 nerve root** and the **S1 nerve root.** The space has a volume of 1–2 mL.

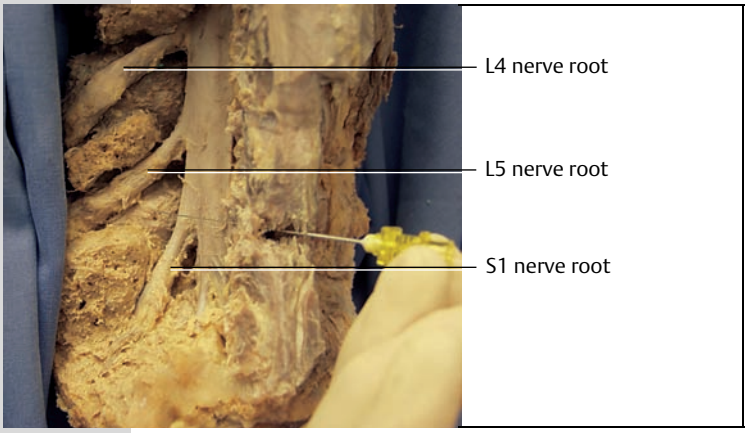


Fig. 9.179 Lumbar spine specimen from the left lateral/posterior view. Needle position, directed toward the L5/S1 intervertebral disk between the L5 and S1 nerve root, demonstrated on an anatomical specimen.

Fig. 9.180 Lumbar spine specimen from the left lateral/posterior view. The fine 29 G cannula is found in the triangle formed by the L5 and S1 nerve roots and the upper edge of the sacrum.

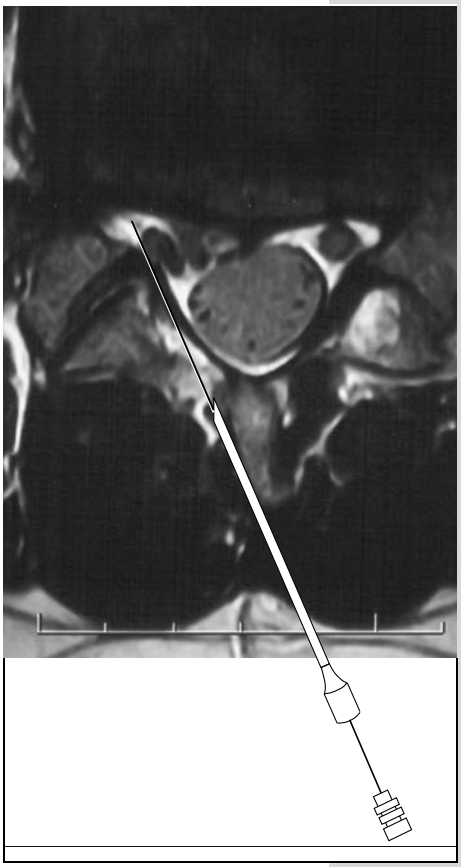


Fig. 9.181 Final needle position illustrated on an MRI scan.

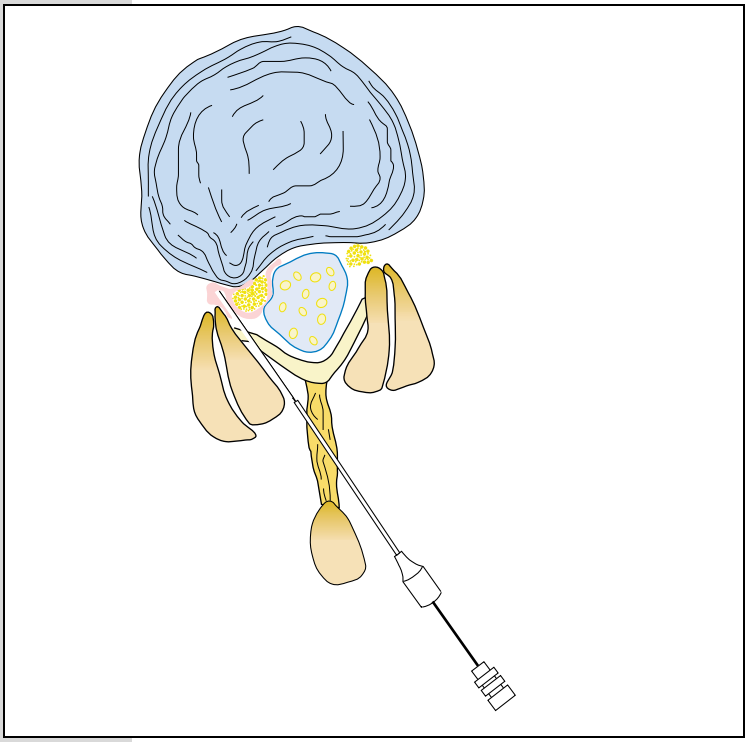


Fig. 9.182 Flooding of the right nerve root, which is modified by inflammation and located directly adjacent to an intervertebral disk protrusion.

Special Situations during the Epidural Perineural Injection (Figs. 9.183–9.191)

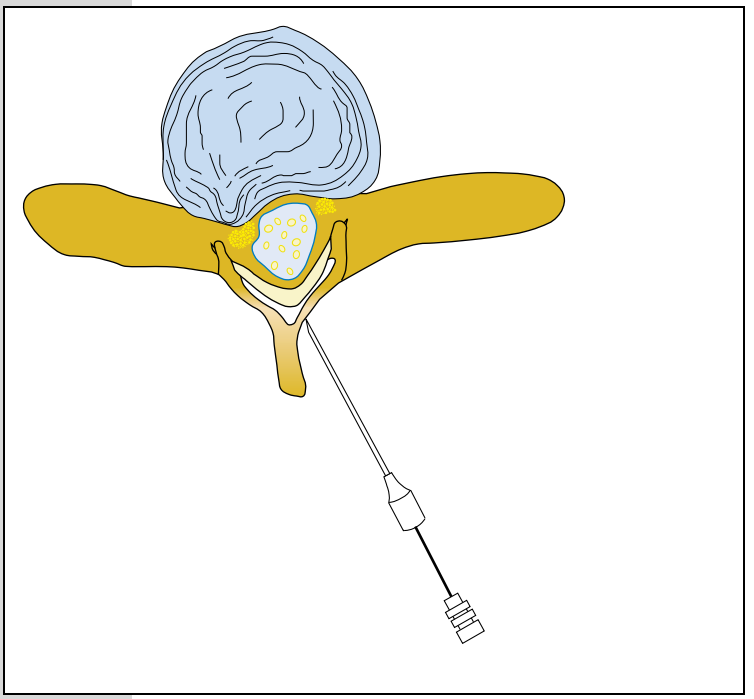


Fig. 9.183 **Special situation I:** Contact with the vertebral arch. This form of contact is felt very early on, sometimes even with the introducer cannula. By changing the insertion angle in a superior/inferior direction, i. e., by raising or lowering the end of the cannula, it is possible to distinguish the edge of the vertebral arch and then insert the needle further. When the angle in the horizontal plane is too great, it is impossible to avoid inserting the needle again.

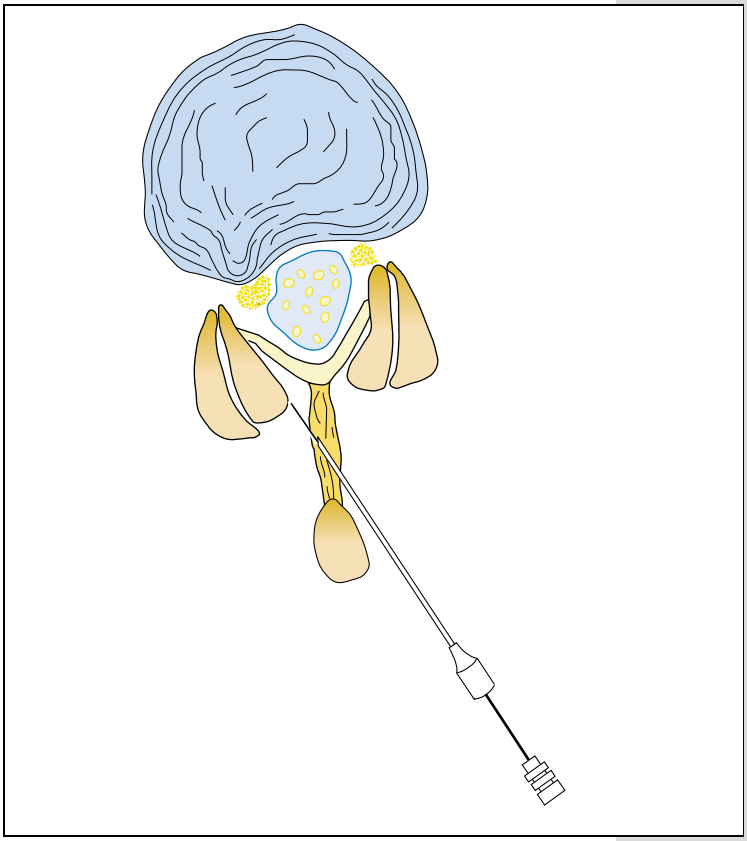


Fig. 9.184 Special situation II: Immediate contact is made with bone (the medial section of the facet) when the 29 G needle is passed through the introducer cannula.

Fig. 9.185 Special situation II, demonstrated on an anatomical specimen: The 29 G needle has made premature bony contact with the medial section of the facet after passing through the introducer cannula. An entrance to the epidural space can be found by:
1. Withdrawing the 29 G needle back into the introducer cannula
2. Reducing the angle of insertion in the horizontal plane
3. Inserting the 29 G needle in a deep position.
If the same bony contact is made again, procedures 1–3 are repeated once more. When facets protrude widely, the angle of insertion is reduced to less than 15°, sometimes less than 10°.

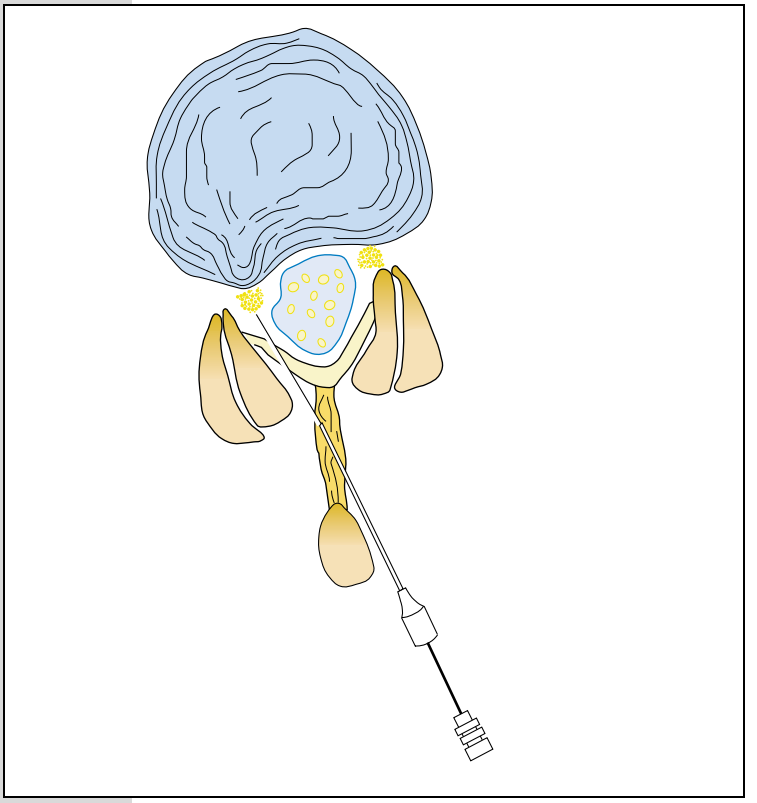


Fig. 9.186 Special situation III: The patient indicates feeling a lightning-like pain spreading into the leg as the 29 G needle is inserted. Patients should be warned of this possibility when the introducer cannula is inserted, at the latest, explaining that they may possibly feel something in their leg. Although the pain is not extreme, it should nevertheless be mentioned beforehand. The target tissue has been reached when nerve root contact is made and the patient experiences their typical form of radiation into the leg. It makes no difference whether the nerve root is flooded from anterior or posterior.

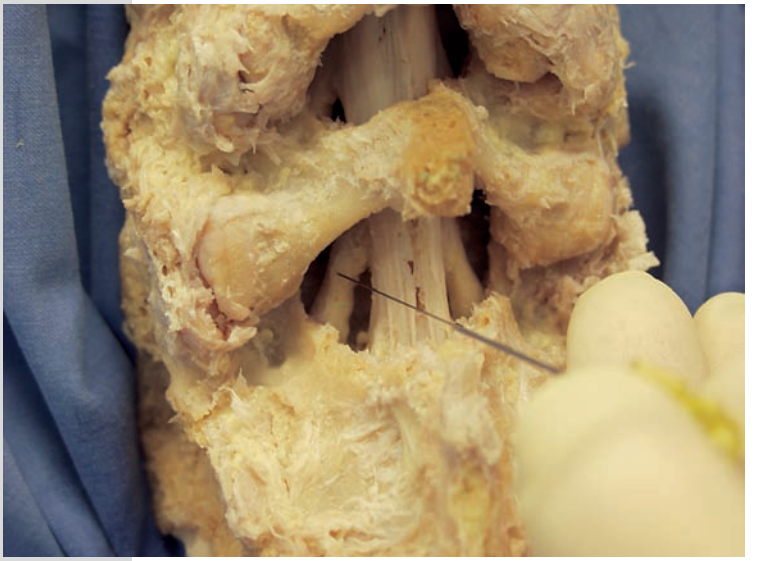


Fig. 9.187 Special situation III, demonstrated on an anatomical specimen: Nerve root contact (without bony contact). The needle is withdrawn somewhat, as every further manipulation of the needle and injection of fluid causes the patient more pain. It is however necessary to aspirate before injecting as the needle tip may be located intrathecally. Injection is possible when the needle is inserted slightly further without renewed sensations of pain and the needle tip is positioned so that it is free of cerebrospinal fluid. The position of the needle must be altered if this is not possible.

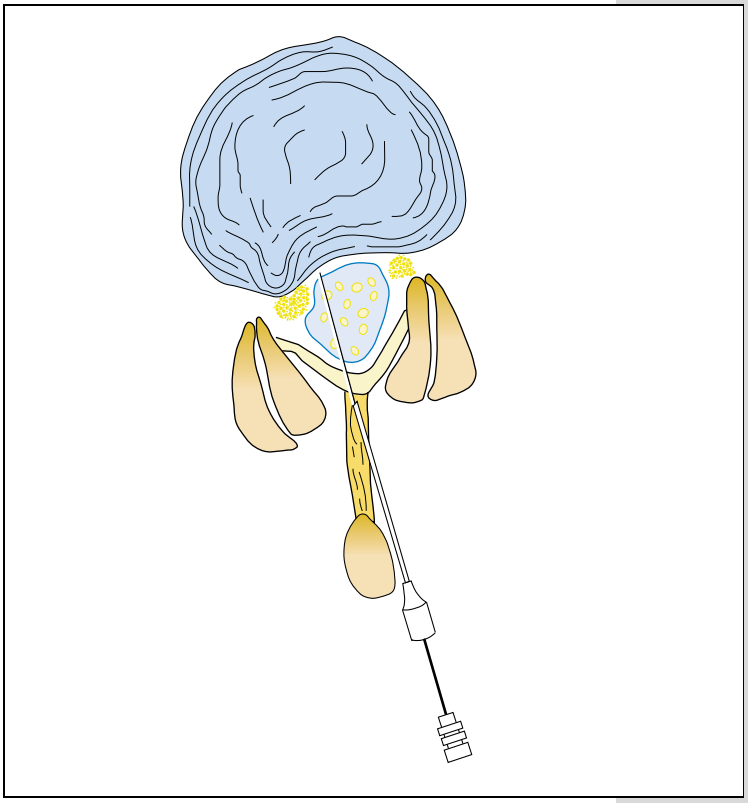


Fig. 9.188 Special situation IV: Transdural needle path. It is to be expected that the needle will pass through the dural sac when targeting the anterolateral epidural space and the angle of insertion is small (following bony contact with the facet when the insertion angle is normal) and the dural sac is wide. Transdural needle paths normally occur with epidural perineural infiltrations in the higher segments.

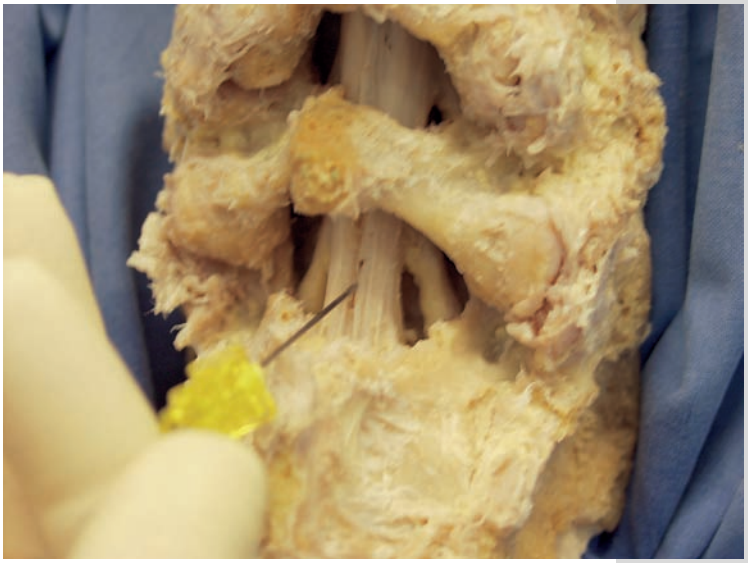


Fig. 9.189 Special situation IV, demonstrated on an anatomical specimen: Transdural needle path with a 29 G cannula.

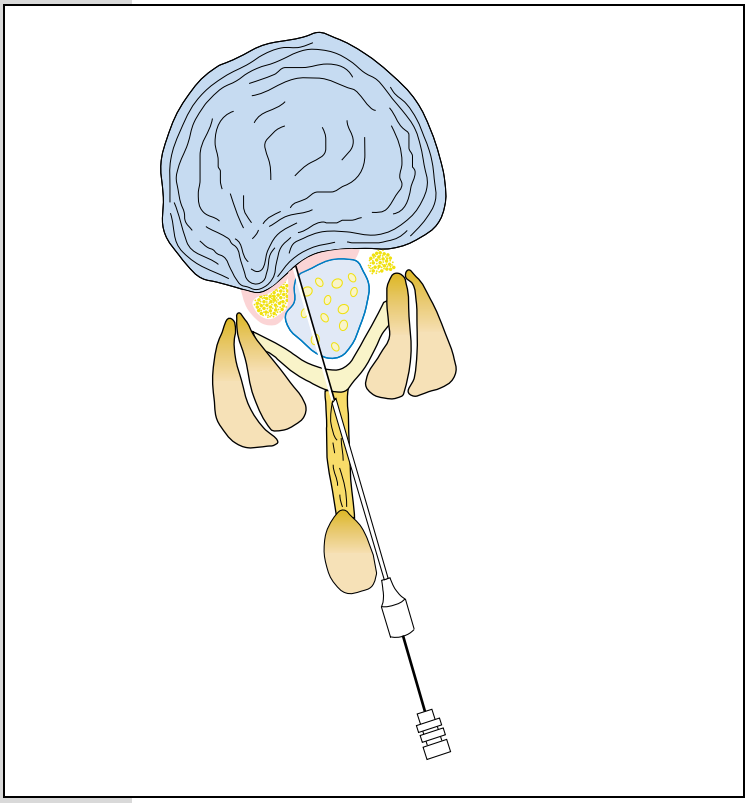


Fig. 9.190 Special situation IV: The spread of local anesthetic and anti-inflammatory in the anterolateral epidural space after passing through the dural sac.

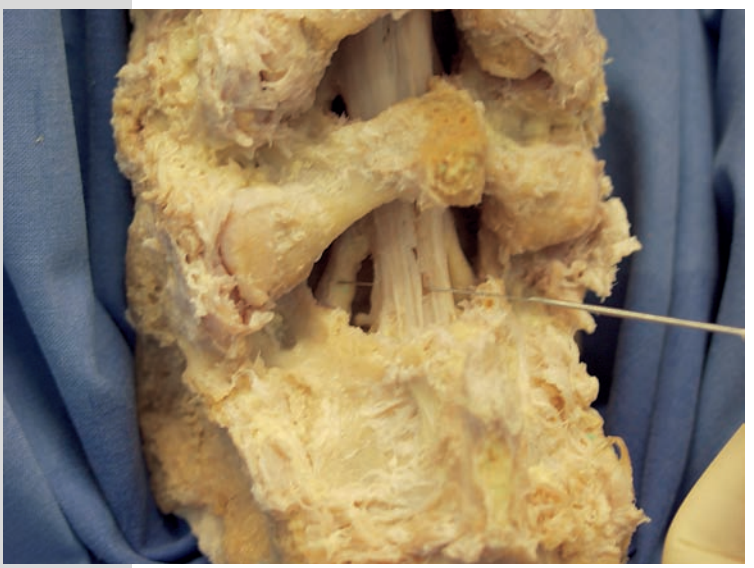


Fig. 9.191 Special situation V, demonstrated on an anatomical specimen: Transdural needle path with the tip of the needle lateral to the S1 nerve root.

Epidural Perineural Injection Using Radiographic Monitoring (Figs. 9.192–9.196)

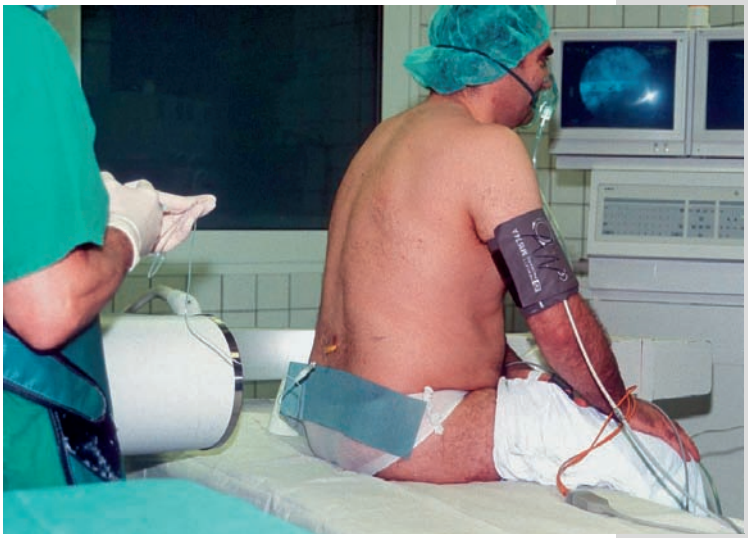


Fig. 9.192 The patient sits during the radiographically monitored epidural perineural injection. The physician places the needle system according to the palpatory anatomical orientation points and then takes an anteroposterior or lateral radiograph. Contrast agent is injected via a connecting tube while the imaging is in progress.

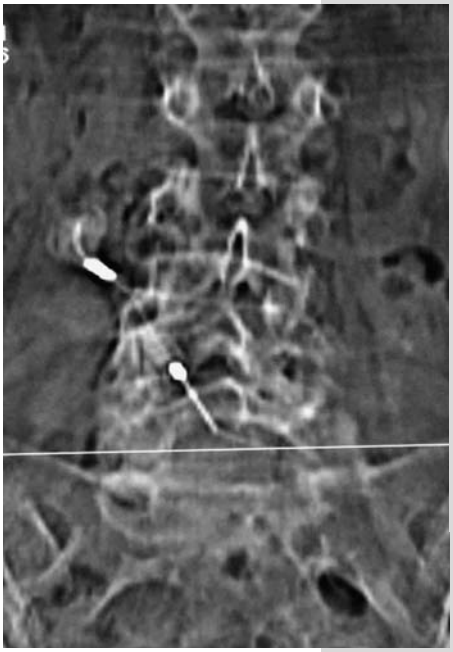


Fig. 9.193 After checking the needle tip position and injecting contrast agent with epidural spread, anti-inflammatories are infiltrated into the epidural space (without image monitoring).

Fig. 9.194 Epidural perineural injection with CT. The needle system is inserted and the angle corrected under CT monitoring. The tip of the needle is located in the anterolateral epidural space. As well as the aspiration of cerebrospinal fluid when withdrawing the needle, this image of the dural path is also added to the documentation.

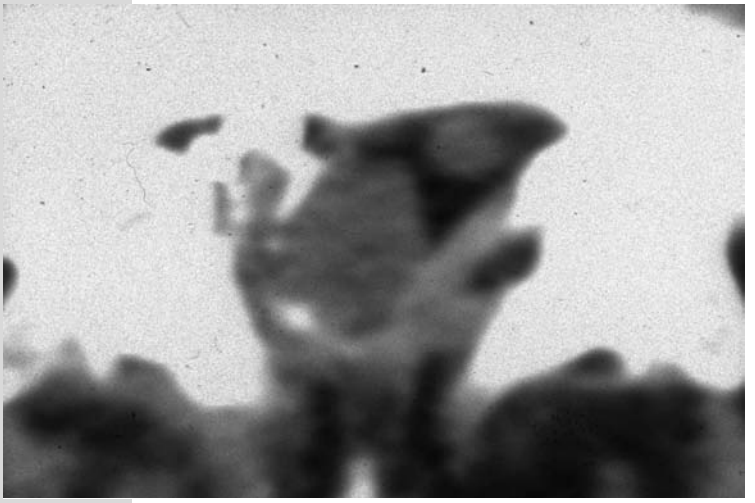


Fig. 9.195 A CT scan of nerve root flooding using a contrast agent.

Fig. 9.196 The patient lies on one side following the epidural perineural injection. The patient should lie on the affected side while resting after treatment or activity on the day of the injection and, if necessary, on the following day. This ensures that the depot injection administered into the triangle stays there as far as possible, so that it can act in that location.

10 General and Specific Complications and Treatment Measures

Vasovagal Syncope

Vasovagal syncope and orthostatic collapse are associated mainly with a drop in blood pressure, tinnitus, pallor, nausea, and in some cases, short-term clouding of consciousness or loss of consciousness. The symptoms are mostly harmless and quickly reversible. Anxiety and sometimes state of sobriety also often play a role.

Treatment

Placing the patient in a horizontal position and general measures, such as calming the patient, generally improve symptoms. Vasovagal reactions often occur during the first session in a series of local anesthetic treatments (Hanefeld et al. 2005). It is nevertheless necessary to consider more serious causes (see below) as a differential diagnosis.

Intravascular Administration of Local Anesthetics and Glucocorticoids

Generally speaking, free local anesthetic that is not bound to proteins interacts with all electrically excitable membranes following its spread into the plasma, blocking the highly specific sodium channels of the membrane. Depending on the concentration of the local anesthetic, all excitable cell systems may be affected.

Other than the local tissue toxicity (nerves, muscle) of some agents, or methemoglobinemia following prilocaine administration, the main side effects of local anesthetics affect the central venous and cardiovascular systems (**Table 10.1**). When the substance-specific limit value of the local anesthetic is exceeded following accidental intravenous injection and overdose, or unexpectedly rapid resorption, symptoms arise that are associated with an increase in plasma concentration of the free substance.

Table 10.1 Toxicity of Local Anesthetics

Systemic toxicity	Effects on CNS Cardiovascular
Local tissue toxicity	Neurotoxicity Myotoxicity
Hematological toxicity	Methemoglobin production (prilocaine)
Anaphylactoid reaction	Monoester type LA >>> Amino amide type

The development of clinical symptoms is highly dependent on the speed of uptake, the plasma concentration, and the type of local anesthetic chosen. Injections administered into the arteries leading to the brain (vertebral artery, carotid artery) result in sudden and sometimes extremely high concentrations in the CNS, with immediate symptoms. The injection of local anesthetics into peripheral arteries or veins results in a comparatively slower uptake.

Central Nervous System

The initial symptoms are those of CNS hyperexcitability; the symptoms of CNS depression develop later (**Table 10.2**). The symptoms may increase gradually, or may attain a high level immediately.

Hypoventilation results in respiratory acidosis (CO_2 retention) and sometimes hypoxia-related metabolic acidosis. In this case, the local anesthetic is released from the plasma protein and the amount of free, active local anesthetic in the plasma increases. More anesthetic can be found in the CNS as a result of the increased hypercapnic brain circulation, and it accumulates in the brain because ions are trapped in their active form in acidotic cells. This in turn accelerates the vicious cycle of symptoms. The risk is generally greatest with highly potent and long-acting local anesthetics such as bupivacaine and ropivacaine.

Table 10.2 CNS Toxicity of Local Anesthetics (Stages, Symptoms, Neurophysiological Correlates)

Stage		*Symptoms*	*Neurophysiology*
1	Prodomal stage	Perioral numbness, tingles, taste disorder (tinfoil) Hyperacusis Anxiety, panic	Only partially direct effects on CNS
2	Preconvulsion stage	Tremor, coordination disorders Tinnitus, reduction in visual acuity Nystagmus Somnolence	Blocks inhibitory neurons in the cortex
3	Convulsion stage	Generalized tonic-clonic cramping with apnea and loss of vigilance	Cramping potential: 1. Amygdaloid body 2. Hippocampus
4	CNS depression stage	Coma Apnea Vasomotor failure Bradycardia Hypotension	Blocking of excitatory neurons and centers EEG neutral line (Indirect circulatory involvement)

Treatment

Treatment should follow the guidelines for effective cardiovascular resuscitation, and the necessary expertise and equipment for this must therefore be readily available. The prognosis for CNS symptoms is generally good when they are adequately treated.

Cardiac Circulatory System

The cardiac circulatory system generally seems more resistant to the systemic action of local anesthetics. CNS symptoms typically arise at lower plasma levels than cardiac circulatory symptoms. However, this does not apply to all local anesthetics. There is significantly less difference when using long-acting agents, such as bupivacaine, compared with the mid-acting local anesthetics (lidocaine, mepivacaine, prilocaine). Indirect cardiac circulatory effects via the CNS (bradycardia, arrhythmia, and sympathicolysis) must be differentiated from the direct effects (negatively dromotropic, inotropic, and repressive of the pacemaker function in the sinoatrial nodes). Modern local anesthetics, such as ropivacaine or S-bupivacaine, seem to be better in this respect. **Figure 10.1** shows the symptoms depending on the time and dosage.

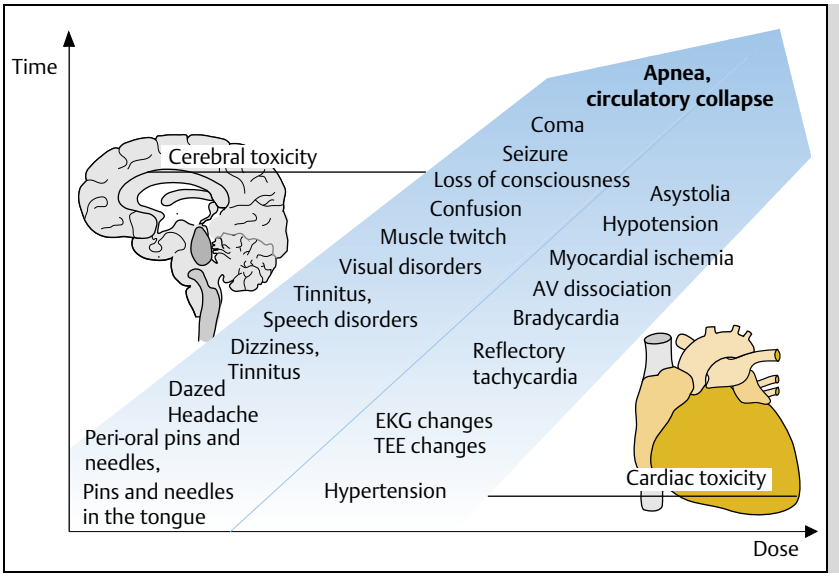


Fig. 10.1 Systemic and toxic symptoms following the administration of local anesthetics.

Treatment

Treatment is limited to symptomatic measures such as the administration of oxygen and, if necessary, artificial respiration, the administration of fluids, and vasopressors if required. If necessary, resuscitation is carried out according to the Advanced Cardiac Life Support (ACLS) standards.

Intrathecal Administration of Local Anesthetics and Glucocorticoids

When local anesthetics are accidentally injected into the subdural or subarachnoid space, the local anesthetic can reach the intracranial region and bind to the central neuronal structures, to an extent depending on the volume and dose. The typical symptoms arising from this are known as "total spinal anesthesia" (classically arising from an overdose of intrathecal local anesthetic during spinal anesthesia or from unnoticed intrathecal injection of local anesthetic during peridural anesthesia; **Table 10.3**).

The risk is higher during anesthetic interventions near the spinal cord, and typically during paravertebral, intercostal, stellate, celiac, and thoracic and abdominal sympathetic ganglia blocks. Total spinal anesthesia has even been described following ophthalmological and ENT blocks.

Table 10.3 Symptoms of "Total Spinal Anesthesia"

Coma
Dilated, unreactive pupils
Central apnea
Arterial hypotension (vasomotor failure) up to cardiovascular arrest

Treatment

Treatment is symptom-related. Emergency equipment must be kept available throughout the procedure (**Table 10.4**).

When resuscitation according to the ACLS guidelines is carried out immediately, and when complications such as aspiration, hypoventilation, and/or hypoxia are prevented, the prognosis that the central block will recede is good (depending on the dose and type of local anesthetic administered).

Table 10.4 Treatment for Intrathecal Local Anesthetic

Stop further administration of local anesthetic
Free airways, administer oxygen, artificial respiration, intubate
Support cardiovascular system
Intravenous access
Rapid, bold administration of fluids (e. g., balanced electrolyte solutions) + hydroxyethyl starch (e. g., 6 % HES 130/0.4)
Catecholamine administration (e. g., norepinephrine or epinephrine 0.5–1 mg IV)

NOTE

The intrathecal application of glucocorticoids is not known to have any acute life-threatening side effects.

Anaphylactoid Reaction—Anaphylactic Shock

Anaphylactoid reactions are immunological or paraimmunological reactions associated with the release of typical mediators—serotonin, slow-reacting substance of anaphylaxis (SRS-A), bradykinin, arachidonic acid metabolites, platelet activating factor, and histamine. These reactions should be considered as potentially life-threatening, and require rapid and adequate treatment.

The clinical reaction is to be expected within 30 min of exposure to allergens; sometimes it may occur immediately. The severity of the reaction is inversely proportional to the latency time. Severe reactions can lead to cardiovascular arrest without any prior warning.

Anaphylactoid reactions are clinically divided into five levels of severity (**Table 10.5**). These levels are not based on the pathological mechanism of the original reaction. The symptoms range from trivial skin efflorescence, mild to severe respiratory and cardiovascular symptoms, or smooth muscle spasms (in the hollow viscera), to immediate and sudden respiratory and cardiac arrest. Symptoms can potentially begin at any level of severity and then subside, persist, or increase.

Table 10.5 Anaphylactoid Reactions: Levels of Severity and Treatment

Level	*Stage*	*Symptoms*	*Treatment*
0	Local (at point of contact with the antigen)	Cutaneous reaction, locally restricted	Stop antigens; if necessary, with H1/H2 blocker
I	Mild general reaction	Disseminated cutaneous reaction (flush, urticaria, pruritis), mucosal reaction (nose, conjunctivitis), general reactions (agitation, headache, mild fever reaction)	Additional prophylactic H1/H2 blocker with wide-bore venous access
II	Distinct general reaction	Measurable, but not life-threatening cardiovascular dysregulation (tachycardia, hypotension), respiratory disorders (mild dyspnea, bronchospasm), gastrointestinal disorders (nausea, urge to urinate and pass stool)	Additional oxygen 6 l/min Glucocorticoid (prednisolone 250 mg) Balanced electrolyte solution, if need be (HES 0.5–2 l) Epinephrine in some cases (0.05–0.2 mg; diluted) Call emergency team
III	Life-threatening general reaction	Shock (severe hypotension, pallor), life-threatening smooth muscle spasms (bronchi, uterus, intestines, bladder, etc.), clouding or loss of consciousness	Additional epinephrine (0.1–0.5 mg) Energetic administration of fluids (HES, small-volume resuscitation if needed) Intubation, cricothyrotomy if needed Call emergency team
IV	Vital organ failure	Cardiovascular arrest and/or respiratory arrest	**Cardiopulmonary resuscitation** following the ACLS guidelines, epinephrine (3 mg in 10 mL saline endobrachial or 0.5–1 mg in a fast-running infusion)

Treatment

The initial treatment (see **Table 10.5**) consists of stopping the influx of allergens immediately. Obviously, this is not possible after the injection of a local anesthetic.

Even when symptoms are mild, IV access should be obtained right away and kept open by using a large-bore needle for the infusion of a balanced electrolyte solution. Oxygen should be administered prophylactically (this is obligatory for more severe reactions). It is advisable to administer histamine-receptor blocking agents, e. g., dimethpyrindene (Fenistil) 4 mg and cimetidine (Tagamet) 200 mg, via the IV line. However, their action is not likely to be visible within the first 30 minutes. The same applies to glucocorticoids (e. g., Solu-Decortin H 250 mg).

If there is a significant drop in blood pressure, the energetic administration of fluids is indicated, e. g., a pressure infusion of hydroxyethyl starch (HES 130/0.4, 6 %, 500–2000 mL). When there is a marked drop in respiration rate, the infusion of hyperosmolar colloid (e. g., HyperHAES at a maximum dose of 4 mL/kg body weight) may be considered.

The IV administration of epinephrine (0.05–0.2 mg; ampules up to 1 mg, diluted 1:10) is indicated in all reactions from level 2 upwards. This has an **immediate** vasopressor (α-effect), brocholytic (β-effect), and specific antiallergic effect. The patient must be immediately intubated and, if necessary, resuscitated according to the ACLS standards when respiratory failure (level 3) or respiratory and cardiac arrest occur.

In some cases, swelling of the pharyngeal–laryngeal mucosa may be so great that a cricothyrotomy is required to obtain an artificial airway. Laryngeal obstruction is the most common cause of death in cases of anaphylaxis. It is therefore important to pay attention right away to the symptom of "lump in the throat" (Tryba et al. 1994, Madler et al. 1998, Hofmann et al. 2001).

As far as emergency tactics go, if a resuscitation team is available, it is preferable to alarm them at an early stage. From level 1 reactions onward, the patient should be referred to a hospital emergency department. A high degree of caution is recommended, because of the unpredictability and possibly rapid development of presenting symptoms. Regrettably, there is no consensus between specialties with regard to the prophylactic provisions of expert personnel, equipment, and safety levels.

NOTE

Anaphylactoid reactions may in principle always arise following the administration of local anesthetics.

This often involves the so-called para-group allergies, which are most frequent with the ester type of local anesthetic. Acid amides can, however, also elicit anaphylactoid reactions at all levels of severity. It is especially important

to be wary of local anesthetics supplied in puncturable vials, as these are often mixed with the stabilizer methylparaben, a para-group substance with high allergic potential. This naturally negates the advantages of the acid amides. It is for this reason that glass ampules are preferred.

NOTE

Luckily, allergic reactions to local anesthetics are seldom seen nowadays.

Postdural Puncture Syndrome

The postdural puncture syndrome (PDPS) is characterized by the following symptoms:

- The development of or an increase in headache within 15 minutes of the patient assuming an upright position, accompanied by one or more of the following associated symptoms: stiff neck, tinnitus, altered hearing, photophobia, nausea.
- Previous (un)intended dural puncture.
- The headache develops within 5 days following dural puncture.
- The headache improves within minutes when the patient lies down.

Spontaneous remission of symptoms occurs within 5 days in over 80 % of all cases. The frequency and extent of symptoms is positively influenced by the gauge size of the needle used, the needle type (Quincke > pencil-point needle), the puncture technique (number of trials), gender (f:m = 2:1), the patient's age (highest frequency at 18–30 years), weight (slim patients), previously recurring headaches, and previous PDPS.

From the pathophysiological point of view, PDPS probably arises as a result of a prolonged loss of cerebrospinal fluid from a persistent dural leak. This leads to low intracranial cerebrospinal fluid pressure. The headache arises from the intracerebral venodilation and the mechanical irritation of the meninges as the brain is displaced in a caudal direction.

The diagnosis is generally made using subjective assessment. Additional investigations (cranial CT, MRI) are necessary only in special cases (suspected infection, bleeding, hygroma).

Preventive measures apply only to intentional dural punctures (primarily "nontrauma-inducing" needles, small gauge size, inserting the mandrin again before removing the needle, and rubbing parallel to the longitudinal dural fibers when using trauma-inducing needles). Prophylactic bed rest is useless.

Treatment

The patient will be happy to follow the order for bed rest when symptoms are present. The following is also recommended for **mild, short-term symptoms**:

- caffeine p. o. (3 × 200 to 4 × 300 mg per day), or
- theophylline p. o. (3 × 350 mg per day).

For **strong pain of longer duration:**

- intravenous caffeine (500 mg, slowly)
- epidural autologous blood patch (20 mL at the same level as the puncture, afterward 2 hours lying prone; 85 % success rate after the first injection, 98 % after the second injection).

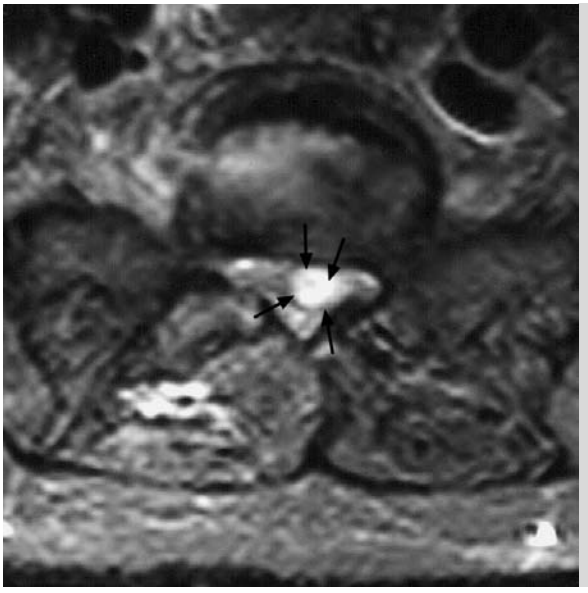
The use of increased administration of fluids, abdominal pressure, or epidural saline infusions is controversial.

Bacterial Infection

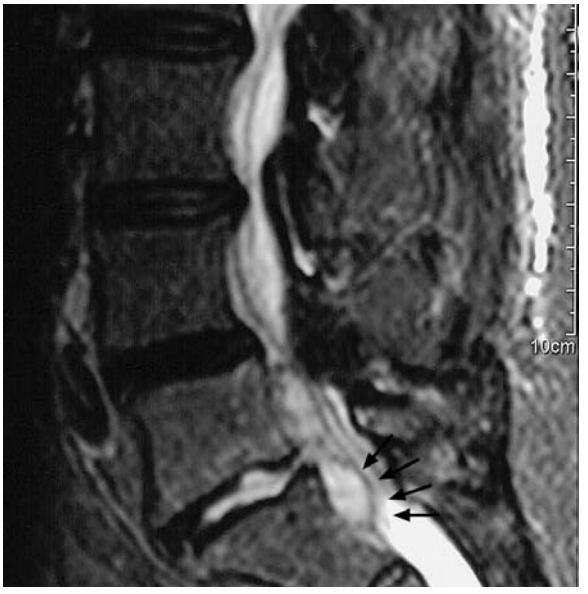
The risk of infection is significantly higher when injecting glucocorticoids near the spine than it is for a joint puncture. It is essential to follow strict guidelines, and the rules of asepsis must be correctly adhered to. An analysis by Bernau (1994) indicates that the risk of infection is 1 in 35 000 injections.

The use of spinal injection therapy is contra-indicated when bacterial infections are already present or when the patient is suffering from a systemic infection. When planning regular injection therapy over a long treatment period, it is therefore wise to monitor inflammatory parameters (CRP, leukocytes) by laboratory testing before starting the injection series and at regular intervals during the period of treatment.

The development of clinical symptoms of inflammation—skin redness, rise in temperature, and pain on pressure at the area of the injection—justifies the suspicion that a bacterial infection has arisen as a complication. The clinical examination involves the assessment of neurological status, motor activity, and sensation to ascertain the presence of meningeal involvement. Antibiotic treatment is commenced immediately at the highest possible dose; it is initially administered intravenously, in order to reach a constant effective concentration. Also, it is always neces-



a



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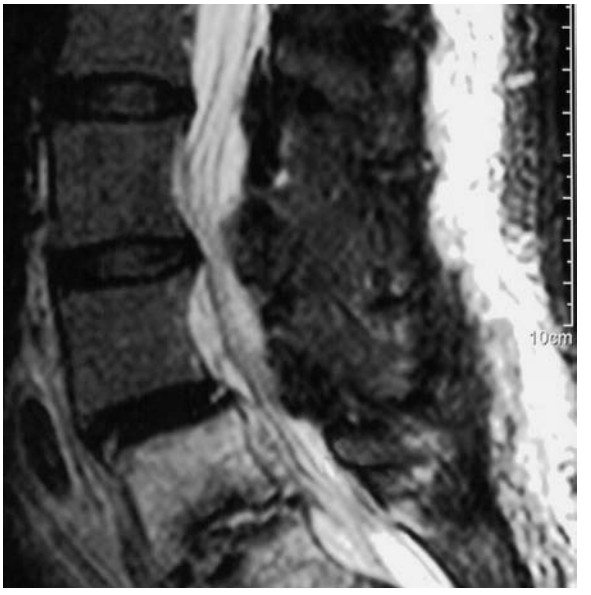


c

Fig. 10.2a–c MRI of epidural abscess in the left L5/S1 segment. Transverse plane (**a**) and sagittal plane (**b**) (arrow) following previous injection therapy (bilateral CT-guided facet infiltrations at L5/S1 carried out five times by office-based physician). Emergency surgery to relieve the abscess followed. Two months later a spondylitis/spondylodiscitis developed at the same segment (**c**).

sary to assess whether surgical intervention is indicated. A further MRI scan of the affected vertebral segment is carried out as soon as possible to diagnose the presence of an epidural abscess before it is too late (**Fig. 10.2a–c**). It has been proven that staphylococci are frequently found in infections following injections. Until an antibiogram is available, a bactericidal antibiotic that is effective against staphylococci should therefore be the drug of choice. Appropriate drugs that enter the cerebrospinal fluid are isoxazolyl penicillins, such as oxacillin or flucloxacillin, and the second- and third-generation cephalosporins. When the relevant symptoms are present, blood cultures must be taken to prove the presence of bacteria and to prepare an antibiogram. The interventions required to gain sample material for microbiological examination must not, however, be allowed to delay the start of the antibiotic therapy. A change of antibiotic may be necessary once the results of the antibiogram are available.

NOTE

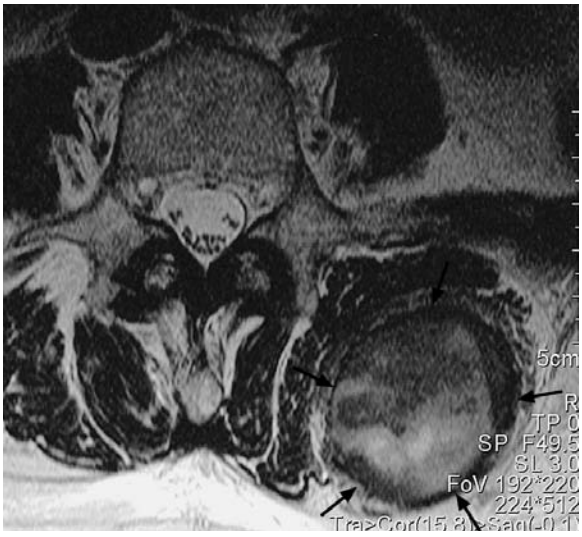
It is always necessary to assess whether surgical intervention is required. When a proven epidural abscess is present, surgical intervention is usually necessary to open the abscess and drain the cavity. The aim is to prevent the spread of infection into the intraspinal space. Epidural abscesses following single-shot injections are rare when the asepsis guidelines are followed.

Bleeding

The injection of medication should not be continued if bleeding is seen to occur during the procedure. A new injection should be planned, on the following day at the earliest, and at a different injection site. The injection site where the bleeding occurred should be thoroughly clinically assessed before starting on further injections. In cases of unusual hematoma formation, the injection therapy should not be continued until a bleeding diathesis has been excluded.

The use of sonography or MRI is advisable when bleeding has occurred near the spine. These investigations enable exact localization and assessment of hematoma size (**Fig. 10.3a–d**). Clinically checking the neurological status is obligatory. In rare cases, surgical dissection is necessary when bleeding causes nerve root compression or when very large hematomas are present.

a



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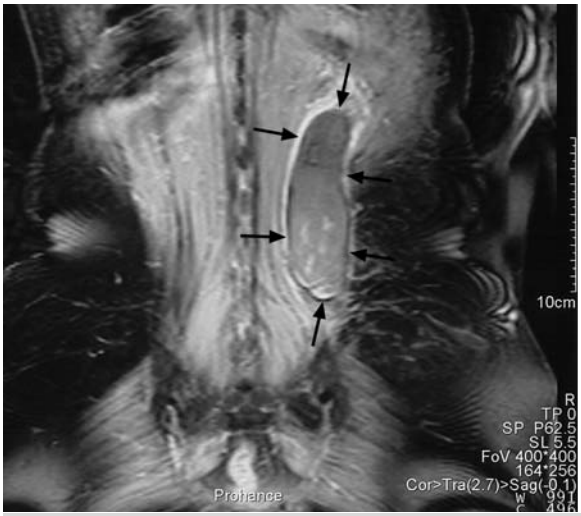

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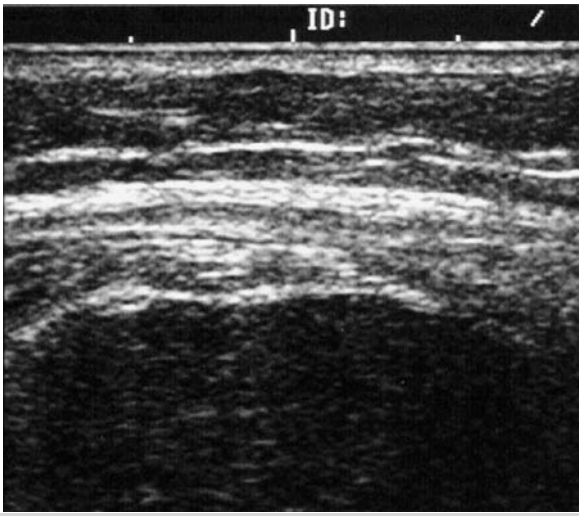


d

Fig. 10.3a–d The clinical picture (**a**) of a left paravertebral hematoma following a LSPA injection. The patient did not mention taking aspirin (ASS 300) on admission or when the medical history was taken. MRI showing the magnitude of the hematoma: Transverse (**b**) and entire views (**c**) (arrows). The hematoma was therapeutically relieved under sonographic monitoring (**d**). The increasing pain symptoms were then immediately relieved.

Special Complications and Side Effects of Cervical Spinal Nerve Analgesia (CSPA)

Orthostatic circulatory reactions, dizziness, and drug intolerance are general risks for every type of injection (see above) and are treated according to their clinical symptoms. Recent investigations (Hanefeld et al. 2005) have demonstrated that observed cardiovascular complications of CSPA are most likely to be associated with benign vasovagal presyncope. The measured hemodynamic parameters (blood pressure, pulse rate, oxygen saturation, respiratory rate) did not show any statistically significant changes during the observation period. Cardiac dysrhythmias did not occur.

The patient will usually perceive a feeling of warmth in the ipsilateral arm and more rarely in the affected side of the body within minutes following the CSPA. This is a typical concomitant symptom and not a complication. This feeling can remain for several hours, depending on the duration of effect of the local anesthetic used.

Stellate Block

The unwanted but not life-threatening side effects of CSPA include **stellate ganglion blocks**. The stellate ganglion is the most inferior of the three sympathetic ganglia. It is located on the prevertebral cervical fascia at the same level as the seventh cervical vertebra, between the base of the transverse process and the neck of the first rib. When a sufficient concentration of the local anesthetic disperses into the ganglion, a temporary one-sided stellate block occurs with the formation of a **Horner syndrome** and the associated **miosis, ptosis, and enophthalmos**. This particularly occurs with CSPA at C6/7 and C7/T1. The cervical sympathetic chain supplies the heart, carotid body, pharynx, and thyroid gland, but these organs are only likely to be affected by a double-sided stellate block. The injection treatment should therefore be planned so that a bilateral block is safely avoided.

In advance of the planned outpatient CSPA injection therapy patients must be informed that, in cases of doubt, **on the day of the injection they will not be fit to drive after the treatment**, allowing them to prepare for this eventuality.

Pneumothorax

Accidental damage to the pulmonary pleura may result in a pneumothorax. This relatively serious complication occurs with a probability of 1–1.5 % (Kattapuram et al. 1992, Krämer and Nentwig 1999). In most cases puncturing the tip of the lung with a cannula does not result in a pneumothorax, as the small leak caused almost always closes up immediately. However, emphysema cavities or lung bullae at the perforation site can cause a continual flow of air in the pleural space and result in a pneumothorax (**Fig. 10.4**). The clinical signs of injury to the pleura can include stabbing pain during inhalation and exhalation, respiratory distress, and the urge to cough. A pneumothorax resulting from an injection injury develops more slowly than one resulting from a penetrating injury to the thorax. The full clinical range of symptoms may therefore not be seen until several days later. A clinical examination of the affected patients shows the following major symptoms: A hypersonorous percussion sound, diminished respiratory sounds compared with the other side, and decreased movement of the affected side of the thorax when breathing. The absence of cyanosis does not exclude a pneumothorax. A patient who is suffering from respiratory symptoms following the injection should therefore be kept under medical care until the symptoms have improved. **The use of a pulse oximeter during the injection increases the chance of early recognition of gas exchange disorders** caused by a pneumothorax (Helm et al. 1997).

Treatment

A radiographic examination of the thorax during expiration must be conducted immediately when the presence of a pneumothorax is suspected. A minor pneumothorax can be treated with hospitalization and watchful waiting. The accumulated air will spontaneously resorb over a period of several days when no further air exits. In all other cases, the positioning of a thoracic drain (**Fig. 10.5**) in the second intercostal space along the mediolavicular line is necessary. The tip of the drain should be located at the dome of the pleura (Glinz 1979).

Nerve Root Sleeve Injection

Injecting into the sleeve of the nerve root, i. e., an endodural injection, is not something to be feared when the injection is done in a technically correct manner. Anatomical studies on specimens have proved that the nerve root sleeve never protrudes over the bony structures (Krämer 2009). The temporary development of quadriplegia has been described (Karasek and Bogduk 2004) when the technique has been modified (Leriche, Fontaine, and Herget blocks; **Fig. 10.6**) and the local anesthetic diffuses into the intrathecal space. Accidental damage to the vertebral artery is not anatomically possible when the technique is employed correctly (posterior CSPA technique according to Reischauer).

Fig. 10.4 Pneumothorax on the right following a CSPA at C7/T1 where the needle was incorrectly positioned.

Fig. 10.5 Thoracic drainage and complete recovery of the lung.

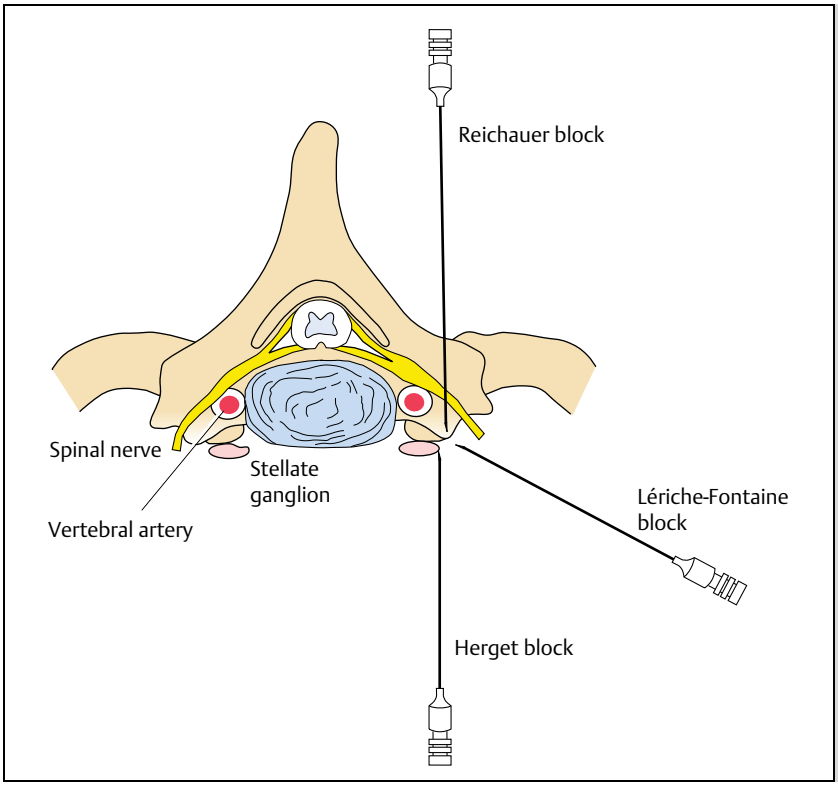


Fig. 10.6 Topography of the stellate ganglion and the different possibilities available for injection: posterior, anterolateral, and anterior access.

Side Effects and Complications of Cervical Epidural Injections

Infections are particularly to be feared because of the location of the administration site. As well as strictly following the rules of asepsis, it is important to look out for skin impurities, local inflammation, and infected sebaceous glands. When these precautions are observed, the development of epidural abscesses is rare (Huang et al. 2004).

Dural Puncture

Accidental dural puncture with **postdural puncture headache** is possible. As with accidental lumbar dural punctures, the therapy consists of lying flat, analgesics, and ensuring adequate fluid intake. This may take the form of infusions in some cases (see above, "Postdural Puncture Syndrome"). Accidental intrathecal injection of corticoids can be prevented by routinely conducting epidurograms to confirm the needle position beforehand. When the contrast agent is seen to be intrathecal, no medication is administered. The intrathecal injection of medication is treated along the same lines as dural puncture injury, i. e. , bed rest for 24 hours and the administration of fluids. There is no possibility of causal therapy.

Side Effects and Complications of Thoracic Injection Techniques

The most important complication arising from thoracic spinal nerve analgesia and the infiltration treatment of costotransverse joints is iatrogenic injury to the lungs and the pneumothorax associated with this (see above). The same rules apply as when treating a **pneumothorax** caused by cervical injections. This complication is rare when the correct injection technique is used (two-finger protective technique).

Side Effects and Complications of Lumbar Spinal Nerve Analgesia

One typical side effect that often occurs a few minutes after the paravertebral lumbar spinal nerve analgesia (LSPA) is a **subjective feeling of warmth in the back and the ipsilateral leg**, which occurs in conjunction with a reduction in back and leg pain. It should not be seen as a complication. The anesthetizing of sensory nerve fibers leads to a temporary **numbness** in the affected leg. Our observations indicate that approximately **8 %** of local anesthetic blocks on motor nerve fibers result in **motor disorders**. These are caused by the medication diffusing into the epidural space via the intervertebral foramen and so reaching the neighboring nerve roots. The patients concerned complain of difficulties walking and standing: A typical comment from patients is that the leg "gives way." Patients must be informed of this side effect before the injection, to prevent falls. Before commencing treatment it should be ascertained that the patient has access to any necessary help, especially when the injection is conducted in an outpatient setting.

Severe cardiovascular reactions, such as cardiac arrest, are not to be expected after a single injection of 10 mL or less of dilute local anesthetic. We have not experienced this type of complication with more than 100 000 paravertebral injections over the past 10 years. It is not necessary to routinely obtain intravenous access, use EKG monitoring, or have a defibrillator readily available.

Temporary **vasovagal circulatory reactions occur in 4.3 %** of patients. Young patients with a history of orthostatic dysregulation are predisposed to this. Older patients with previously existing cardiovascular disorders are not endangered by LSPA. High blood pressure, coronary heart disease, and dysrhythmias are not negatively affected (Hanefeld et al. 2005).

Spinal Anesthesia

In very rare cases, nerve root sleeve injections (see above) can result in complete or **incomplete spinal anesthesia with motor disturbances in both legs**. In these cases it is necessary to raise the upper body, and give the patient an intravenous infusion of electrolyte solution. The patient must be kept under clinical observation until the spinal anesthesia has resolved. In some cases it may be necessary to monitor the patient using EKG and use a pulse oximeter to measure oxygen saturation (see "Intrathecal Administration of Local Anesthetics and Glucocorticoids," above). This complication occurs with an **incidence of approximately 0.3 %** (Krämer 2009). Because of damage to the

dura, the resulting development of low cerebrospinal fluid pressure with postdural puncture headache is a possibility. It is therefore recommended that patients maintain bed rest over the following days, lying flat for 24 hours, and that suitable quantities of fluids are administered (see "Postdural Puncture Syndrome" above).

Kidney Puncture

A further complication, when needles are badly misplaced, is an **injury to the kidney**. Suspected kidneys injuries should be checked using **sonography,** and a **urine analysis** should be obtained. Investigations following needle biopsies of the kidneys demonstrated an increased risk of arteriovenous fistula development caused by the puncture (Lozano et al. 1978). When there is a suspicion that the **renal pelvis has been punctured**, **further specialized urological** treatment is indicated in the case of permanent leakage.

Pure stab wounds to the kidney parenchyma generally do not require further treatment. In contrast, **renal capsule hematomas** may require **surgical dissection**. This last-mentioned complication has occurred only once in our clinic and was most likely due to an incorrect needle position. No further secondary damage occurred following surgical dissection.

A paravertebral abscess that had to be opened and drained was described as a **serious complication** in our clinic. No further secondary damage occurred following surgical dissection in this case either.

Side Effects and Complications of Lumbar Epidural Injections

Dural Puncture

In addition to the same side effects and complications as are found in LSPA, lumbar epidural injections can accidentally be administered intrathecally when the injection is performed incorrectly. Serious side effects are nevertheless not to be expected: Intrathecal administration of cortisone crystal suspensions is common in the neurological treatment of certain diseases.

A postdural puncture headache can occur when using interlaminar access with the needle passing through the dura before it reaches the anterolateral epidural space. Using the epidural perineural double-needle technique recommended here with a **29 G cannula**, postdural puncture headache will arise, for example, in < 1 % of all cases when the needle passes through the dura. In these cases, only a moderate level of pain is to be expected. Bed rest is generally not required, but lying flat, analgesia, and adequate fluid intake for the following 24 hours are recommended.

Recent investigations (Willburger et al. 2005) into cervical and lumbar spine injection treatment have shown that when the injections are conducted in the manner described here, the **risk of complications is low**. A total of 7963 injections were divided into cervical and lumbar spinal analgesia; facet infiltrations; lumbar epidural perineural, posterior epidural, and sacral epidural injections; and sacroiliac joint injections. Of these, 25 cases (0.3 %) displayed unwanted side effects. Postdural puncture headache occurred in 10 cases following epidural perineural injections and in 3 cases following lumbar spinal nerve analgesia. Five patients experienced circulatory dysregulation with dizziness, nausea, and a drop in blood pressure following epidural perineural injections. One patient fell following a epidural perineural injection and one patient reported a sensory block up to T6 after lumbar spinal nerve analgesia. Local allergic reactions at the injection site occurred in five patients after mepivacaine was injected. All complications were effectively treated symptomatically and were without serious consequences.

Summary

Despite the possible side effects and complications, spinal injection therapy is one of the safest and most effective methods used in pain therapy when it is done correctly. It is not necessary to routinely obtain intravenous access or use EKG monitoring. Nevertheless, EKG equipment and a medical emergency case with an intubation set and the corresponding medication should be available nearby, and the practice employees should be trained in emergency treatment. More literature on special complications of spinal injections can be found in Kraemer (2009).

11 Multimodal Spinal Therapy

The spectrum of treatment for back and neck pain ranges from the simple application of heat and administration of analgesics at one extreme to open surgery at the other. Multimodal spinal therapy is in the center of this range. It deals primarily with nerve root compression, treating it with a combination of local injections, physical therapy, pain treatment, and behavioral training. The severity of the presenting symptoms determines whether the multimodal program is conducted on an outpatient basis in a day clinic or in an in-patient setting. A multimodal intensive program should be followed when there is no compelling reason for immediate surgery. This program aims to improve symptoms quickly and permanently, preventing chronification and the need for possible surgery later on as a result of ineffective conservative treatment.

Outpatient Minimally Invasive Spinal Therapy

Multimodal spinal therapy, including spinal injections, can in principle be carried out on an outpatient or day hospital basis. Apart from the few interventions that require 24-hour monitoring, such as cervical epidural injections, all the special interventions conducted by physicians can be administered in an outpatient setting. **Table 11.5** on page 213 shows the limiting factors for outpatient treatment, which are also the indications for in-patient treatment.

Patients who are admitted to hospital usually present with severe pain, a considerable maladaptive posture, and paralyses that are in the gray area of becoming indications for surgical intervention. They usually arrive at the hospital by ambulance as they are otherwise unable to travel. In-patient observation is also necessary in cases where large prolapses are present, as there is a risk of further paralysis. **The indication for the outpatient treatment of a nerve root compression syndrome is therefore "diagnosis by exclusion."**

The first day of outpatient treatment consists of the usual detailed examinations and determining the diagnosis. Once the further diagnostics with radiography and, in some cases, laboratory findings are completed and the patient has received a sufficient amount of information, adequate pain therapy should be administered on the first day in the form of spinal nerve analgesia or epidural injection. In acute cases with severe pain, the cervical or lumbar spinal nerve analgesia is conducted daily over the following days. This is followed in each case by physical therapy, thermotherapy, Glisson traction, Fowler position or side-lying for the lumbar spine, and electrotherapy.

Cervicobrachial syndromes and lumboischialgia have a strong tendency to become chronic. For this reason, pain coping strategies are introduced right from the start. Progressive muscle relaxation follows during the consecutive sessions. The invasive interventions are reduced, with intervals of 2–3 days depending on how the symptoms develop.

Epidural perineural injections and other types of epidural injection therapy are administered a maximum of three times over the entire treatment cycle, with a break of several days between injections. Depending on which is the most prominent primary and secondary pain as time goes on, trigger point and facet infiltration, sacroiliac joint infiltrations, acupuncture, or other interventions drawn from the wide spectrum of treatment possibilities can be administered. In some cases, concomitant therapy with medication is necessary right from the start (see Chapter 4, "Multimodal Medication Concomitant Therapy"; **Tables 11.1, 11.2**).

The patient should also see the physician again some time after the third week, e. g., after a further 3–6 weeks, depending on the amount of irritation in the cervical or lumbar nerve root. The physician uses this opportunity to assess the patient's orthopedic and neurological status and check the diagnosis. When necessary, local infiltrations are conducted to desensitize the nerve root at this point in time and later at increasingly longer intervals.

The remaining components of the multimodal program are carried out by patients themselves. This applies especially to exercises commencing in the pain-relieving position, and sports that are gentle on the spine.

Table 11.1 Outpatient Pain Therapy Concept for Lumbar Nerve Root Syndrome Injections

1st week	*1st day (Mon.)*	*2nd day (Tue.)*	*3rd day (Wed.)*	*4th day (Thur.)*	*5th day (Fri.)*	*6th day (Sat.)*	*7th day (Sun.)*
	Adm	LSPA	Epi	FAC	LSPA	LSPA	Fow
	Ro	Fow	SL	Fow	Fow	Fow	TT
	Laboratory	ET	TT	ET	TT	TT	
	Info	PT	PT	PT	PT		
	LSPA	Int.PCS	PMR	PMR	PMR		
	Fow						

2nd week	*8th day (Mon.)*	*9th day (Tue.)*	*10th day (Wed.)*	*11th day (Thur.)*	*12th day (Fri.)*	*13th day (Sat.)*	*14th day (Sun.)*
	Epi	ACU	LSPA	SIJ	ACU	SIJ	Fow
	SL	Fow	Fow	Fow	Fow	Fow	TT
	TT	PT	ET	TT	PT	TT	
	PT	PMR	PT	PT	PMR		
			PMR	PMR			

3rd week	*15th day (Mon.)*	*16th day (Tue.)*	*17th day (Wed.)*	*18th day (Thur.)*	*19th day (Fri.)*	*20th day (Sat.)*	*21st day (Sun.)*
	ACU	PT	LSPA	PT	ACU	Fow	Fow
	Fow	PMR	Fow	PMR	Fow		
			ET				

Abbreviations:

ACU	acupuncture
Adm	admission
BS	back school
CSPA	cervical spinal nerve analgesia
Disch	discharge
Epi	epidural injection
ET	electrotherapy
FAC	facet infiltration
Fow	Fowler position
Glis	Glisson traction
GrPT	group physical therapy
Info	patient information
Int.PCS	pain coping strategies
Laboratory	laboratory findings
LSPA	lumbar spinal nerve analgesia
PMR	progressive muscle relaxation
PT	physical therapy
Ro	radiography
SIJ	sacroiliac joint infiltrations
SL	side-lying
TP	trigger point infiltration
TT	thermotherapy.

Table 11.2 Outpatient Pain Therapy Concept for Cervical Nerve Root Syndrome Injections

1st week	*1st day (Mon.)*	*2nd day (Tue.)*	*3rd day (Wed.)*	*4th day (Thur.)*	*5th day (Fri.)*	*6th day (Sat.)*	*7th day (Sun.)*
	Adm	CSPA	FAC	ACU	CSPA	FAC	ACU
	Ro	Glis	Glis	Glis	Glis	Glis	Glis
	Laboratory	ET	TT	ET	TT	ET	TT
	Info	PT	PT	PT	PT		
	CSPA	Int.PCS	PMR	PMR	PMR		
	Glis						
2nd week	***8th day (Mon.)***	***9th day (Tue.)***	***10th day (Wed.)***	***11th day (Thur.)***	***12th day (Fri.)***	***13th day (Sat.)***	***14th day (Sun.)***
	ACU	CSPA	Glis	CSPA	ACU	TP	Glis
	Glis	Glis	TT	Glis	Glis	Glis	TT
	ET	TT	PT	TT	ET	TT	
	PT	PT	PMR	PT	PT	PT	
		PMR		PMR	PMR		
3 rd week	***15th day (Mon.)***	***16th day (Tue.)***	***17th day (Wed.)***	***18th day (Thur.)***	***19th day (Fri.)***	***20th day (Sat.)***	***21st day (Sun.)***
	ACU	PT	CSPA	PT	ACU	TT	Glis
	Glis	PMR	Glis	PMR	Glis		
	ET		TT		ET		
	PT		PT		PT		

For abbreviations, see Table 11.1.

In-patient Minimally Invasive Spinal Therapy

Intensive IMIST should be conducted over a period of 5–10 days before operating on spinal symptoms that are extremely resistant to treatment. This does not apply if a serious acute paralysis requires immediate surgery. Most cases involve a nerve root compression syndrome arising from an intervertebral disk prolapse, spinal canal stenosis, or postsurgical scarring. In terms of invasiveness, IMIST is intermediate between outpatient specialist orthopedic treatment and open surgery (Theodoridis and Krämer 2003) (**Table 11.3**). In most cases symptoms improve in the long term, so that open surgery need no longer be considered as a treatment option. The improvement of symptoms is achieved by giving daily injections of analgesics to the spinal nerve and epidural perineural infiltrations, and at the same time implementing a special physical therapy program that continues after the patient is discharged.

The IMIST concept is multimodal, containing medical, physical therapy, and psychotherapy components. The concept has proved itself over the last 20 years with more than 15 000 patients at the Orthopedic University Clinic at St. Josef-Hospital Bochum in Germany. It is continually being improved on the basis of experience and scientific studies. The essential components of the multimodal program— spinal injections, movement therapy, and behavioral training (back school)—are evidence based and specifically recommended by the Drug Commission of the German Medical Association (**Table 11.4**).

Table 11.3 The Treatment of Nerve Root Compression Syndromes

Outpatient	In-patient	In-patient
General practitioner	IMIST	Open surgery
Specialist physician		

Table 11.4 Evidence for Conservative and Minimally Invasive Treatment Methods in Cases of Back/Leg Pain (Krämer 2009, German guidelines back pain 2006)

	Studies	*Evidence*
Epidural injection	9	↑ ↑
LSPA	2	↑
Intradiscal laser	2	↓
Percutaneous nucleotomy	2	↓
Chemonucleolysis	4	↑
Physical therapy	< 6	↑
Back school	18	↑
NSAIDs	25	↑ ↑
Myotonolytic agents	15	↑

Medical Interventions

The medical interventions are supervised by specially trained orthopedists and pain therapists. Following the minimally invasive interventions (injections), the patient is placed in a special pain-relieving position or in Glisson traction. This is individually adjusted and checked by the physician. Further daily medical interventions e. g., peripheral infiltrations, manual therapy, and acupuncture, are performed at other times and depend on the findings.

The patient's self-assessment of pain and the clinical neurological findings are assessed regularly, and pain medication is adjusted individually during the in-patient stay. In special cases, changes in medication are decided upon in an interdisciplinary pain conference involving medical pain specialists, psychotherapists, and internal medicine physicians. The pain medication is also assessed after discharge, in consultation with the patient's general practitioner.

Physical Therapy

The complementary physical therapy consists of
- exercise and muscle strength training
- back school
- thermotherapy
- electrotherapy.

The physical therapy interventions are integrated into the daily routine.

In addition, an individualized sport and movement program is introduced, based on the pain-free range (MIPFR) concept. This continues after the patient has been discharged.

Psychotherapy

These sessions, run by psychologists, take place mainly during the late afternoon. They include training in how to cope with pain and exercises that target muscle relaxation, e. g., Jacobson's muscle relaxation.

The psychologists also introduce the patients to self-help programs, to provide them with a way of coping with their remaining symptoms after they have been discharged.

Special Interventions

Some special diagnostic and therapeutic interventions are also used in special cases alongside the standard IMIST program. These include:
- spinal infiltrations under image guidance (CT or MRI)
- discography and intradiscal therapy
- interlaminar, transforaminal, or sacral epidural endoscopic therapy
- autologous epidural perineural infiltration with interleukin receptor antagonist protein (IRAP) using the patient's own blood (see Chapter 4, "Interleukin-1 Receptor Antagonist Protein")
- interlaminar or epidural catheterization
- plaster cast test to decide whether fusion is appropriate
- radiculography
- individual physical therapy
- instructions in the use of muscle electrostimulation units
- individual psychotherapy.

Other disorders such as labile hypertension, diabetes, gastrointestinal symptoms, and neurological diseases are treated by consulting physicians from other medical specialisms.

Indications for In-patient Minimally Invasive Spinal Therapy

The main indications for IMIST are serious cervical and lumbar nerve root compression syndromes that cannot be adequately treated in an outpatient setting. They are seen especially as a result of intervertebral disk prolapses, spinal canal stenoses, and postdiscotomy syndromes. Often a combination of these causes is present. Further indications are spondylolisthesis (isthmic or degenerative), osteoporotic fractures, and synovial cysts (**Table 11.5**).

The criterion for in-patient treatment of spinal disorders is the level of severity, i. e., severe pain and neurological deficits. However, outpatient treatment from a specialist physician should always precede a hospital stay, in order to demonstrate that it is impossible to treat the patient's symptoms successfully in this way. Outpatient treatment is not useful when pain is regularly graded above 5 on the numeric rating scale (NRS, 0 = no pain, 10 = worst pain imaginable) and when the pain continually increases under loading, e. g., when the patient gets in and out of a car. The spinal nerve root irritation that is reduced by the pain therapy is reactivated when the patient travels home, and this prolongs the healing process.

Neurological findings that are serious enough for surgery to be considered are usually associated with severe pain and must be treated in an in-patient setting (**Table 11.6**) right away. Surgical intervention is required in the presence of cauda symptoms or the acute loss of important functional muscles. The imaging results (radiography, CT, or MRI) should correlate with the clinical symptoms.

Finally, patients must be willing to be treated on an in-patient basis with daily spinal infiltrations (**Table 11.7**). Conservative IMIST in an acute hospital is not indicated when the daily spinal infiltrations cannot be carried out for any one of the reasons mentioned in this table.

Table 11.5 Indications for IMIST in Case of Serious Findings and Pain Rated > 5 on the Numeric Rating Scale (NRS)

Intervertebral disk prolapse
Decompensated spinal canal stenosis
Postsurgical: Scarring, instability (postdiscotomy and postfusion syndromes)
Spondylolisthesis (degenerative and isthmic)
Osteoporotic fracture
Synovial cysts

Table 11.6 Serious Findings as Indication for IMIST

Considerable malpositioning due to sciatica, torticollis
Large prolapse with the threat of paraplegia or cauda equina symptoms
Motor and/or sensory deficits found in the gray area of becoming an indication for surgery

Table 11.7 Contraindications for Minimally Invasive Spinal Therapy

Mild to moderate symptoms
Infections, open wounds
Neurological seizure disorders
Severe conduction defects
Decompensated heart failure
Blood coagulation disorders (anticoagulant medication)
Known hypersensitivity to local anesthetics

Diagnostics Required before Commencing Minimally Invasive Spinal Therapy

The diagnosis must be confirmed before commencing minimally invasive spinal therapy. Warning symptoms should be investigated by taking a detailed medical history, carrying out a clinical neurological assessment, laboratory investigations, and imaging of the affected spinal segment (**Table 11.8**).

A neurologist and a surgeon must be consulted immediately when cauda equina symptoms and acute loss of important muscle function (foot drop) are present. Consultation with a neurologist, including an EMG assessment, is also initially necessary when less severe pareses are present, to set a baseline and monitor progress when prescribing muscle electrostimulation.

Table 11.8 Warning Signs (Red Flags) for Back/Leg Symptoms (German guidelines back pain 2006)

Cauda equina symptoms, foot drop
Irregularities in laboratory findings
Weight loss
Further neurological symptoms
Bone destructions
History of carcinoma
HIV (and other systemic infections)

Table 11.9 Risk Factors for the Development of Chronic Back/Leg Pain (Yellow Flags; Krämer 2009, German guidelines back pain 2006)

Discontent with career
Low occupational qualifications
Inability to cope with psychosocial demands
Emotional impairment (depression, anxiety)
External locus of control
Inappropriate beliefs about illness
Operant factors (primary and secondary gain due to illness)
Heavy smoker
Poor physical condition
Further pain that cannot be explained

In addition to the somatic diagnostics, psychological assessment is also required. In particular, this involves assessing whether the criteria for the evolution of chronic pain (yellow flags) are present. These risk factors for the development of chronic back pain do not necessarily contra-indicate IMIST, but rather indicate the necessity for a special physical therapy and psychotherapy program (**Table 11.9**).

Multimodal Program

Initial Medical Intervention

Spinal injections form the central focus of the multimodal program. The spinal nerve root irritation and the accompanying severe pain should be treated with a daily injection. This mainly takes the form of cervical or lumbar spinal analgesia, but once or twice a week epidural injections using the single-shot method are substituted. These injections should be administered by the treating physician or his/her deputy in a special infiltration room or an operating room and, if possible, in the morning. The reason for this is that most patients are afraid of injections. If major interventions are completed in the morning they are over and done with early in the day, and the other components of the multimodal program can be carried on without psychological stress.

Before commencing the spinal infiltration, the treating physician should assess the patient's current status and enquire as to how the previous day went. In the case of neurological deficits, the nerve function must also be assessed before starting further treatment. During the first medical intervention, further procedures and possible options within the multimodal program are discussed and the discharge date is set. The data obtained from the neurological assessment are added to the medical notes.

At our clinic we have tried and tested the use of specially designed treatment cards. The diagnosis and the multimodal program interventions already carried out are clearly summarized on these cards. Patients carry their cards with them, and each intervention carried out as part of the multimodal program is noted on the card each day. The treatment card is added to the patient's medical notes at the end of treatment.

When possible, a nurse or medical assistant who usually works on the ward during the day should attend the first medical intervention. Instructions for further action—ordering consultations, imaging techniques, laboratory tests, passing on information to the physicians on the ward, etc.—are therefore not only put in writing but are also directly implemented by the assistant. The nurse or assistant is also available to the patient on the ward during the day.

Second Medical Intervention

As part of the IMIST, in addition to the daily cervical or lumbar spinal nerve analgesia, a second infiltration is planned for later in the day. There should be at least 6 hours break between injections. When the patient has mainly severe radicular symptoms arising from the anterior branch of the spinal nerve, this second infiltration can take the form of further spinal nerve analgesia. Usually the second infiltration deals with the secondary symptoms found outside the vertebral motor segment, e. g., injections into the sacroiliac joints, trigger point infiltrations, and zygapophysial joint capsule infiltrations. Acupuncture is an alternative when the patient reacts well to this form of therapy. When indicated, manual therapy is conducted by physicians as part of the daily routine.

The second medical intervention can and should be conducted by a second physician so that the patient can obtain a second opinion and can ask questions if need be.

Positioning

After the spinal infiltrations, positioning the patient to relieve the spinal nerve root is the second essential therapeutic component of the in-patient spinal treatment. The patient is placed in a pain-relieving position or in Glisson traction when cervical syndromes are present between the individual treatment sessions. When the intervertebral disk tissue is displaced but the anulus fibrosus is intact, traction offers a good therapeutic opportunity for tissue to return to the center of the intervertebral disk. Glisson traction reliably achieves this in the cervical spine and the flexion cube does so in the lumbar spine.

The physician adjusts these orthopedic aids individually in relation to the clinical neurological findings. The Glisson traction should pull in the direction of relief, when possible. Manual therapy techniques are used to ascertain the appropriate direction. The Fowler position may increase symptoms in the lumbar spine. In this case, the patient should lie on one side with the hips and knees bent (**Figs. 11.1, 11.2**).

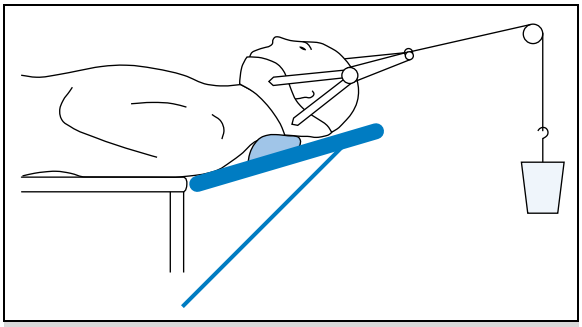


Fig. 11.1 Glisson kyphosis traction applied to the cervical spine and used in causal pain therapy for cervicobrachial syndromes.

Pain Medication

The in-patient care of patients under continual medical supervision also permits the administration of strong analgesics (WHO level 3, see Chapter 4, "WHO Analgesic Ladder") when severe pain is present. However, the aim of the minimally invasive spinal therapy is for patients to get by without pain-relieving medication, or with only small amounts, in conjunction with a long-term movement program. The physician reassesses the medication daily. When problem patients require long-term analgesics, the appropriate pain medication is decided upon in an interdisciplinary pain conference (see Chapter 4, "Multimodal Medication Concomitant Therapy").

Physical Therapy

When physical therapy, thermotherapy, and electrotherapy are used in an in-patient setting, they can be integrated into the daily routine, providing useful supplements to the medical interventions. As the symptoms improve, the physical therapy progresses from exercises based on the pain-relieving position to exercises that are part of the medical training therapy, and the patient starts on an individual sport and movement program.

The movement in pain-free range (MIPFR) concept is based on the observation that movement relieves pain. The prerequisite is that movements should not increase pain, which means that the movements must be made in the parts of the body that are not affected by pain. It is for this reason that the so-called dynamic, "linear" types of sport, such as swimming, jogging, and cycling, are the main focus of the MIPFR program. In the in-patient setting, patients usually start with stationary cycling. This does not normally cause additional symptoms, even in patients with severe spinal pain syndromes.

Prospective randomized studies have demonstrated that movement is better than bed rest when dealing with spinal symptoms (Dietrich 2003, Hildebrand and Pfingsten 1998). The movement caused by suitable exercises and sporting activities stimulates the venous flow in the vertebral canal. The regular alternation between loading and unloading additionally improves the flow of nutrients to the intervertebral disk tissue.

Psychotherapy

Psychologists help in the reduction of permanent pain by providing a special type of training that improves the patient's pain coping skills. This program includes information on how pain is processed and exercises that target muscle relaxation. The daily training takes place in the afternoon.

The most tried and tested method used to train muscle relaxation was developed by Jacobson (1938) and has become widely known as "progressive muscle relaxation." This method teaches patients how to achieve conscious control over tension and relaxation in individual muscle groups step by step. The exercises can be taught in a one-week course during the in-patient stay and continued in the outpatient setting.

The 5–10 Day In-patient Minimally Invasive Spinal Therapy Program

The standard program for in-patient treatment of cervical or lumbar spinal nerve root compression syndromes incorporates a daily minimally invasive medical interven-

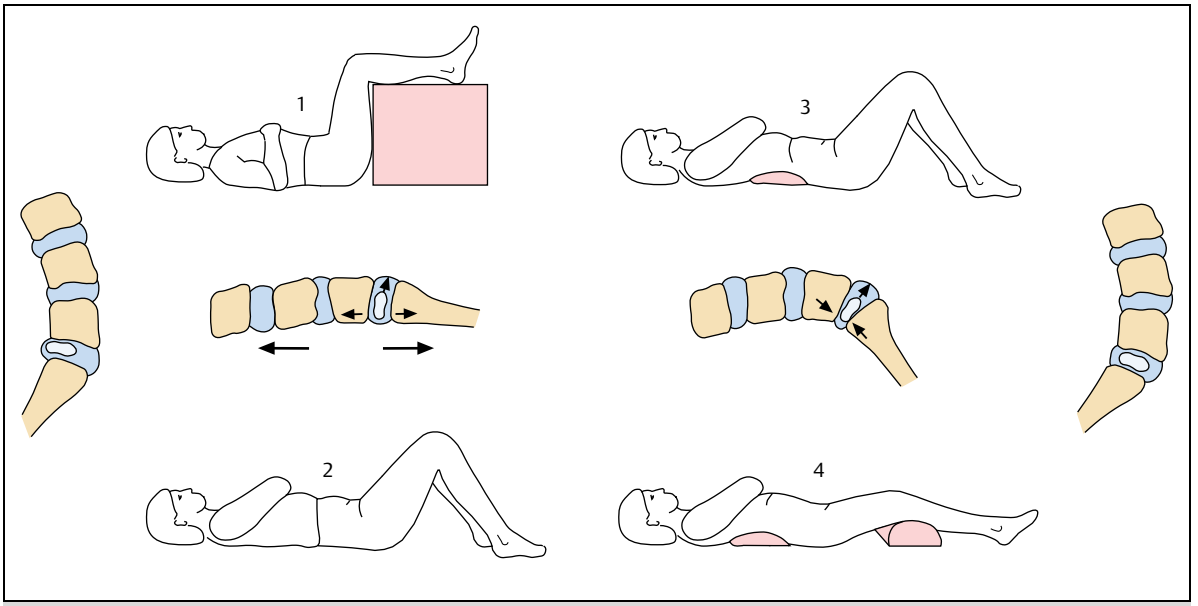


Fig. 11.2 The Fowler position used for radicular lumbar syndromes: Positioning and exercises are initially performed in the pain-relieving position with the hips and knees flexed at 90° (1). As the symptoms decrease with time, the exercises gradually change so that the exercises are increasingly conducted in a physiological lordosis (2–4).

tion in the form of spinal nerve analgesia or an epidural injection. This is commenced after the diagnosis has been confirmed by the initial assessment, the laboratory results, and the findings from imaging. If this information is already available on admission, from the results of outpatient preliminary examinations, a minimally invasive intervention can be commenced on the first day.

The planned standard daily routine for the following days includes placing the patient in the Fowler position or Glisson traction after the spinal injections, depending on which type of injection has been administered. The Fowler position is adopted after spinal nerve analgesia, whereas an epidural injection is followed by positioning in side-lying with flexed hips and knees.

The program is further complemented by individual physical therapy with thermotherapy, electrotherapy, and group physical therapy, as well as postural and behavioral training as part of back school. Depending on circumstances on the ward, afternoon or evenings are used for the second medical intervention. This consists of trigger point infiltration, facet capsule infiltration, sacroiliac joint infiltration, or acupuncture. A further cervical or lumbar spinal nerve analgesia may also be considered as a possible form of second intervention when the radicular symptoms originate primarily in the ventral ramus of the spinal nerve.

The psychologists' work begins with an introduction to pain coping strategies, followed by daily exercises in progressive muscle relation (**Tables 11.10, 11.11**).

The pain arising from a spinal syndrome is generally severe. Medical treatment is therefore also planned for weekends and public holidays, remembering the motto: **"Disk pain never has a day off."** Pain therapy using minimally invasive interventions in the in-patient setting can be conducted by the physician on duty.

Apart from the standard program for cervical and lumbar spinal nerve root compression, treatment may also take another form especially adapted to the presenting symptoms or special types of therapy, e. g., epidural perineural injections using interleukin receptor antagonist protein (IRAP; see Chapter 4, "Interleukin-1 Receptor Antagonist Protein"), catheter treatment, or possible intradiscal diagnostics and therapy. Individual physical therapy or individual psychology sessions are organized in special cases.

Osteoporotic Fractures

The IMIST program is also a possibility for patients with active osteoporosis who have an osteoporotic vertebral fracture. The patient is immediately mobilized as soon as the osteoporotic fracture has been diagnosed. Spinal infiltrations near the vertebral fracture, appropriate pain medication (usually in the form of opioids), and the immediate use of an orthosis enable patients to mobilize sufficiently to use the bathroom facilities with the help of nursing staff, and to eat their meals at a table. The IMIST program takes no more than 10 days, even in the presence of active osteoporosis.

Table 11.10 IMIST Concept for Lumbar Nerve Root Syndrome Injections

1st day (Mon.)	*2nd day (Tue.)*	*3rd day (Wed.)*	*4th day (Thur.)*	*5th day (Fri.)*	*6th day (Sat.)*	*7th day (Sun.)*	*8th day (Mon.)*	*9th day (Tue.)*	*10th day (Wed.)*
Adm	TT	ET	TT	ET	TT	TT	ET	TT	ET
Ro	Epi	LSPA	Epi	LSPA	LSPA	LSPA	Epi	LSPA	TT
Laboratory	SL	Fow	SL	Fow	Fow	Fow	SL	Fow	LSPA
Info	ET	TT	ET	TT	ET	MIPFR	ET	TT	Fow
LSPA	BS	BS	BS	BS	MIPFR	Fow	BS	BS	Info
Fow	GrPT	GrPT	GrPT	GrPT	Fow		GrPT	GrPT	Disch.
	MIPFR	LSPA	MIPFR	MIPFR	Disch?		MIPFR	MIPFR	
	LSPA	Fow	FAC	SIJ			FAC	SIJ	
	Fow	MIPFR	Fow	Fow			Fow	Fow	
	Int.PCS	PMR	PMR	PMR			PMR	PMR	
		ACU		ACU			ACU		

For abbreviations, see Table 11.1.

Table 11.11 IMIST Concept for Cervicobrachial and Cervicocephalic Syndrome Injections

1st day (Mon.)	*2nd day (Tue.)*	*3rd day (Wed.)*	*4th day (Thur.)*	*5th day (Fri.)*	*6th day (Sat.)*	*7th day (Sun.)*	*8th day (Mon.)*	*9th day (Tue.)*	*10th day (Wed.)*
Adm	ET	TT	ET	ET	TT	TT	ET	TT	ET
Ro	CSPA	CESI	CSPA	CSPA	CSPA	CSPA	CESI	CSPA	TT
Laboratory	Glis	Glis	Glis	Glis	Glis	Glis	Glis	Glis	CSPA
Info	TT	ET	TT	TT	ET	ET	TT	ET	Glis
CSPA	BS	BS	BS	BS	MIPFR	MIPFR	BS	BS	Info
Fow	PT	PT	PT	PT	Glis	Glis	PT	PT	Disch.
	CSPA	MIPFR	MIPFR	MIPFR	Disch?		MIPFR	MIPFR	
	Glis	Glis	FAC	TP			Glis	FAC	
	MIPFR	PMR	Glis	Glis			PMR	Glis	
	Int.PCS		PMR	PMR			ACU	PMR	
	ACU		ACU						

For abbreviations, see Table 11.1.

Further Outpatient Treatment

Patients are not free of symptoms when they have completed the 5–10 day intensive in-patient program. Their pain has, however, improved so much that the use of surgery no longer needs to be discussed and a specialist physician or the general practitioner can conduct further treatment in an outpatient setting. The injection therapy is finished. The steroid crystal suspension administered during the epidural injections dissolves gradually and is released slowly, so that a further improvement in symptoms is to be expected following discharge. Paresthesias, weakened reflexes, and motor disorders usually remain after elimination of the main pain. These symptoms generally improve over the following weeks and months if the patient trains appropriately, and normally disappear altogether.

Repeated in-patient stays are not planned as part of the IMIST concept, for two reasons. First, spontaneous remission occurs with discogenic and osseous nerve root com-

pression syndromes. The IMIST accelerates this process by removing the pain peaks. Second, during their hospital stay patients are introduced to an exercise and movement program that has been proven (Dietrich 2003) to stabilize their improvement. Nevertheless, there are always cases where a repeat IMIST is necessary:

- When a new intervertebral disk prolapse or protrusion occurs, combined with nerve root compression.
- When a new decompensated state arises in cases of postdiscotomy syndromes or spinal canal stenosis.

The findings and the indications for surgery must be reassessed when there is a need for repeated in-patient treatment.

Indications for Surgery

In some cases IMIST cannot improve discogenic, osseous, or scar tissue nerve root compression syndromes sufficiently that the patient's quality of life is adequately restored. Even in the absence of absolute indications for surgery, i. e., cauda equina symptoms and paralyses that affect function, surgical decompression or fusion must be considered an option in these cases simply because of the uncontrollable intensity of pain. **Table 11.9** lists the criteria for psychosocial risk factors (yellow flags) which must be excluded.

Severe symptoms demonstrate their persistence even in the first days of IMIST when the patient does not respond to the spinal infiltrations. An open microsurgical intervertebral disk operation is necessary, e. g., when a large prolapse causing severe pain is present and the symptoms do not improve. To avoid endangering the results of the operation (wound healing disorders, cerebrospinal fluid leaks that have not healed), further epidural infiltration of steroids should not be administered in these cases. The decision to operate is generally made after 2–3 days.

In some cases of lumbar spinal canal stenosis, it is not possible to free the patient from his or her decompensated state using minimally invasive spinal therapy and the complementary movement program. An open microdecompression may be considered when pain and restricted walking distance remain unchanged. In this operation only the interlaminar compressing bony and ligamentous structures are removed. This is achieved with a skin incision approximately 2 cm in length and with the use of an operating microscope. In the past, wide laminectomies were common and were sometimes combined with vertebral fusion. This type of operation is not necessary in cases of degenerative spinal canal stenosis (Theodoridis et al. 2005). The indication for microdecompression in cases of lumbar spinal canal stenosis should, however, first be confirmed after discharge from a failed IMIST program because a delayed response to the epidural administration and movement therapy can still be expected.

Postdiscotomy syndromes represent the real problem cases for both outpatient and in-patient treatment. Relapses occur repeatedly, even when the IMIST has been successful. An open surgical neurolysis with fat-grafting surgery may be considered when uncontrollable radicular pain arising from scar tissue is the primary symptom. The so-called plaster cast test is used during in-patient treatment to investigate back pain caused by instability. A plaster cast is applied to the trunk and the affected leg to assess whether the simulated vertebral fusion improves the patient's symptoms.

Patients must be informed that neurolysis and fusion surgery can only improve symptoms, not eliminate them. Patients who have previously undergone multiple operations may, understandably, refuse further open surgery. In this case the only possible treatment is outpatient pain therapy, and periods of in-patient pain therapy with long breaks in between.

Results

Treatment results, mainly obtained from controlled studies, are available for the individual interventions used in the IMIST as well as for the entire program. The effectiveness of epidural injections using glucocorticosteroids in patients suffering from radicular symptoms has been shown in many clinical studies (Jacobson 1938, Klenermann et al. 1984, Cuckler et al. 1985, Griffin et al. 1988, Agency for Health Care Policy and Research 1994, Bogduk 1995, Koes et al. 1995 and 1999, Watts and Silagy 1995, Carette et al. 1997, Krämer et al. 1997a, Hanefeld et al. 2005, Siebertz 2005).

The positive results found in single randomized controlled studies and joint meta-analyses (Van Tulder et al. 1997, McCulloch and Young 1998) based on the data available make an analgesic effect very probable. This statement also largely corresponds to clinical experience (German guidelines back pain 2006).

The standard treatment for cervicobrachialgia and cervicocephalgia is CSPA (Moore 1965, Bogduk 1981, Grifka 1992 and 1996, Fortuna and Fortuna 1993, Bush and Hillier 1996, McQuay and Moore 1998). Rubenthaler et al. (2000) have proved the effectiveness of CSPA in a prospective randomized double-blind study investigating isotonic saline solution vs. local anesthetic.

Two prospective randomized studies have so far investigated the effectiveness of lumbar spinal nerve analgesia

(Krämer et al. 1997a and b). More recent investigations (Ng et al. 2005) have demonstrated the effectiveness of periradicular injections in the lumbar spine in a randomized double-blind study. A significant effect of the injection of bupivacaine and methylprednisolone was demonstrated at the 3 month follow-up. They did not, however, find a statistically significant difference between these two active substances.

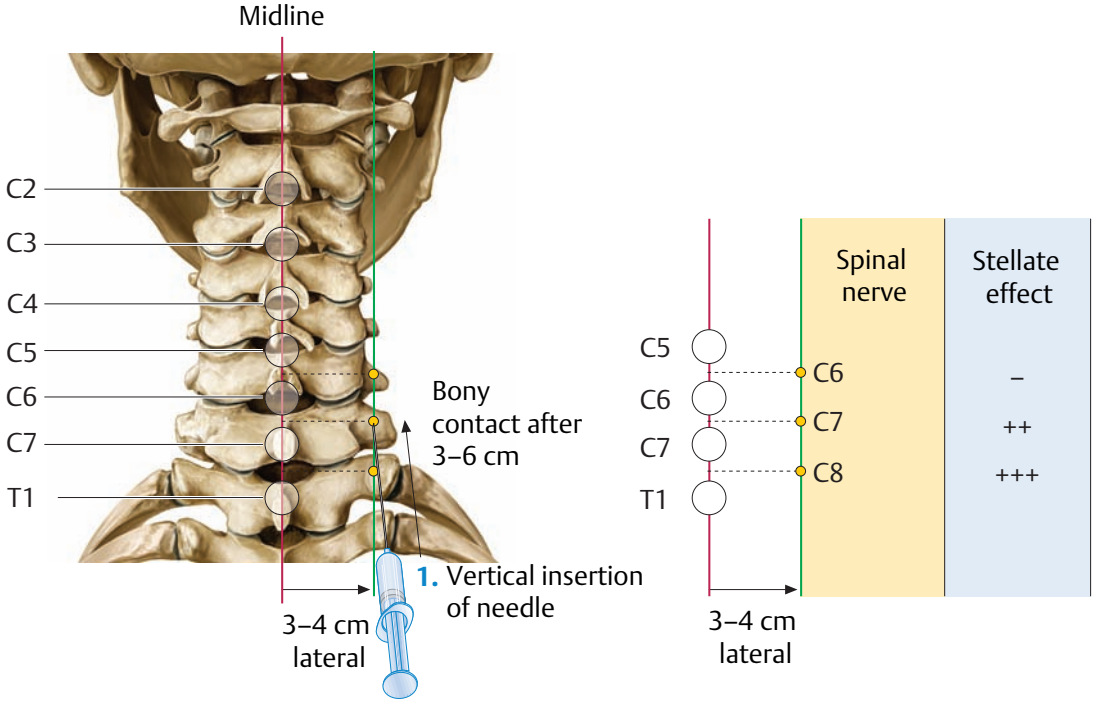
Studies are available that assess the entire IMIST program for the cervical spine and the lumbar spine (Krämer et al. 1997a, Schmidt 2000, Wiese et al. 2001, Lepper 2002, Dietrich 2003, Siebertz 2005). Lepper (2002) conducted a retrospective study into patients who had previously undergone IMIST for cervical nerve root compression. Only one patient of this group had to be operated on in the end. In the case of lumbar nerve root compression, IMIST is so successful that in 92 % of all cases surgery is no longer required (Krämer 1997a). Siebertz (2005) investigated satisfaction levels in patients during treatment and at the conclusion of the program. The results indicated that the total rating for the multimodal therapy program was positive in 91.3 % of patients. This related particularly to the minimally invasive interventions and the second injection. Dietrich (2003) observed at his follow-up assessment 1 year after in-patient treatment that participation in a movement program (MIPFR) resulted in a permanent improvement in symptoms, compared with a control group of patients who did not participate in a movement program.

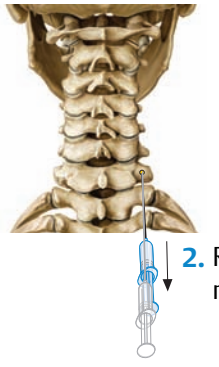
In summary, the results demonstrate that when nerve root compression syndromes in the cervical and lumbar spine are present, attempts should first be made with outpatient treatment or IMIST before confirming the indication for surgery. (More literature on the results of the different spinal injections can be found in Kraemer 2009.)

12 Summary

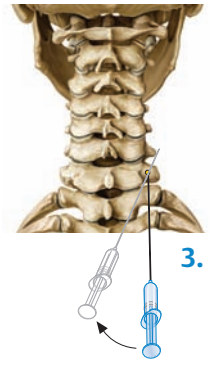
Cervical Spine

CSPA (Cervical Spinal Nerve Analgesia)



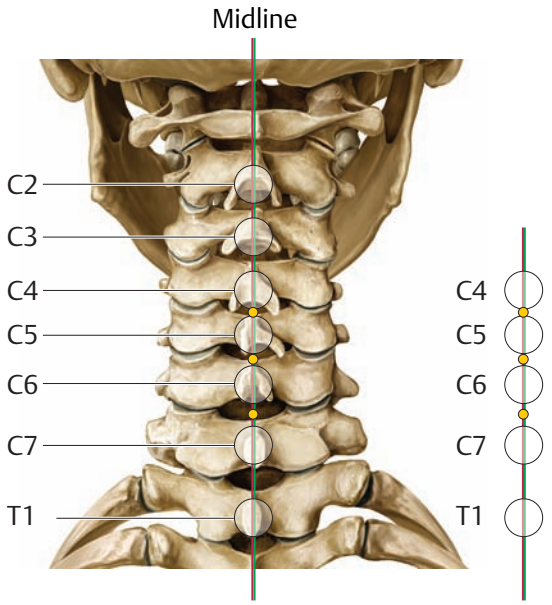


2. Retract needle

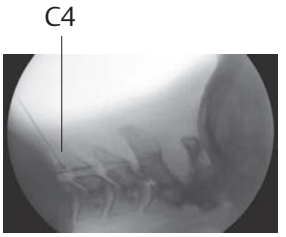


3. Change the needle direction towards craniolateral until the edge of the bone is felt. Insert the cannula **~1 cm** over the edge of the bone.

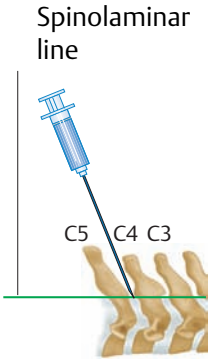
CEI (Cervical Epidural Injection)



1. Injection site is exactly in the midline (C4/C5, C5/C6, or C6/C7)

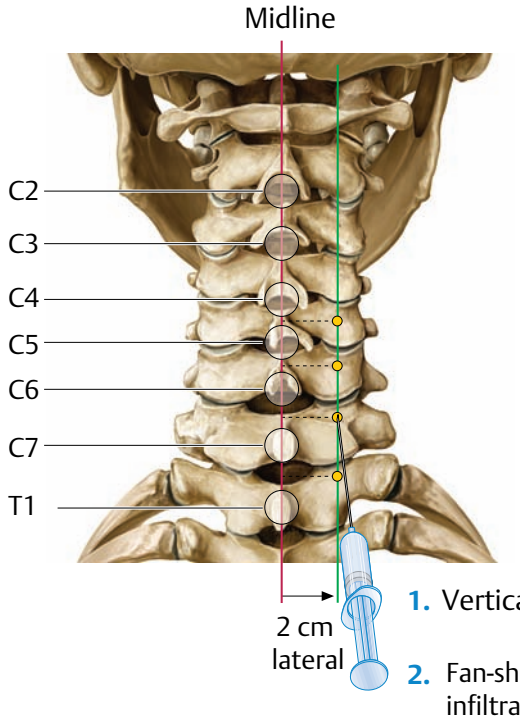


2. Epidurogram (image monitoring, lateral radiograph) when at the same level as the lamina (here C4/C5)



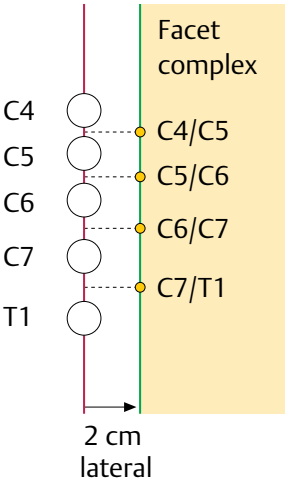
3. Diagram of skeleton, lateral view

CxFI (Cervical Facet Infiltration)



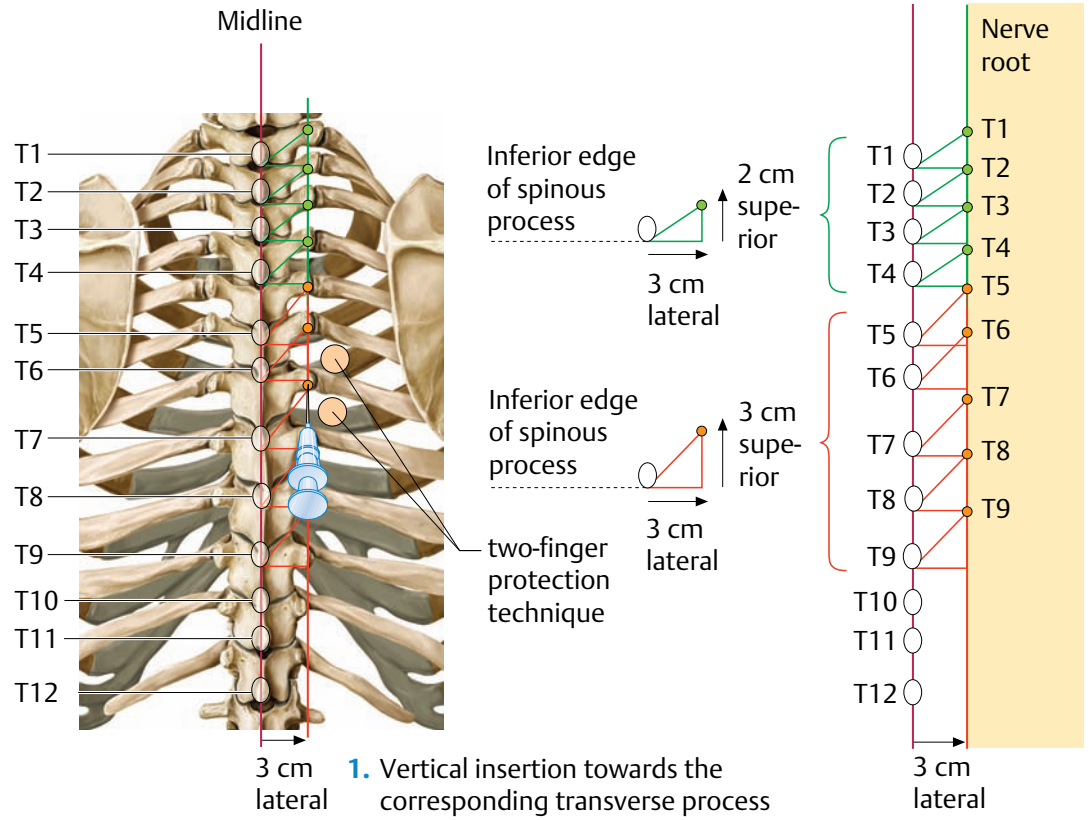
1. Vertical needle insertion

2. Fan-shaped pericapsular infiltration at bony contact

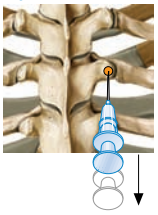


Thoracic Spine

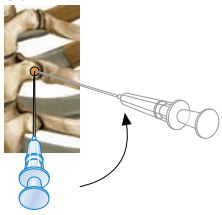
TSPA (Thoracic Spinal Nerve Analgesia)



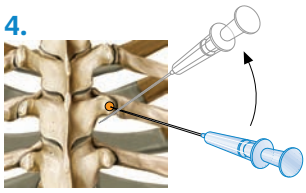
1. Vertical insertion towards the corresponding transverse process



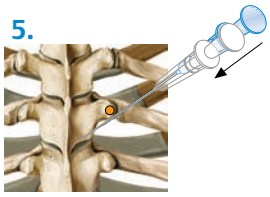
2. Retract until the muscle fascia releases the needle tip



3. Insertion in a 30–40° medial direction

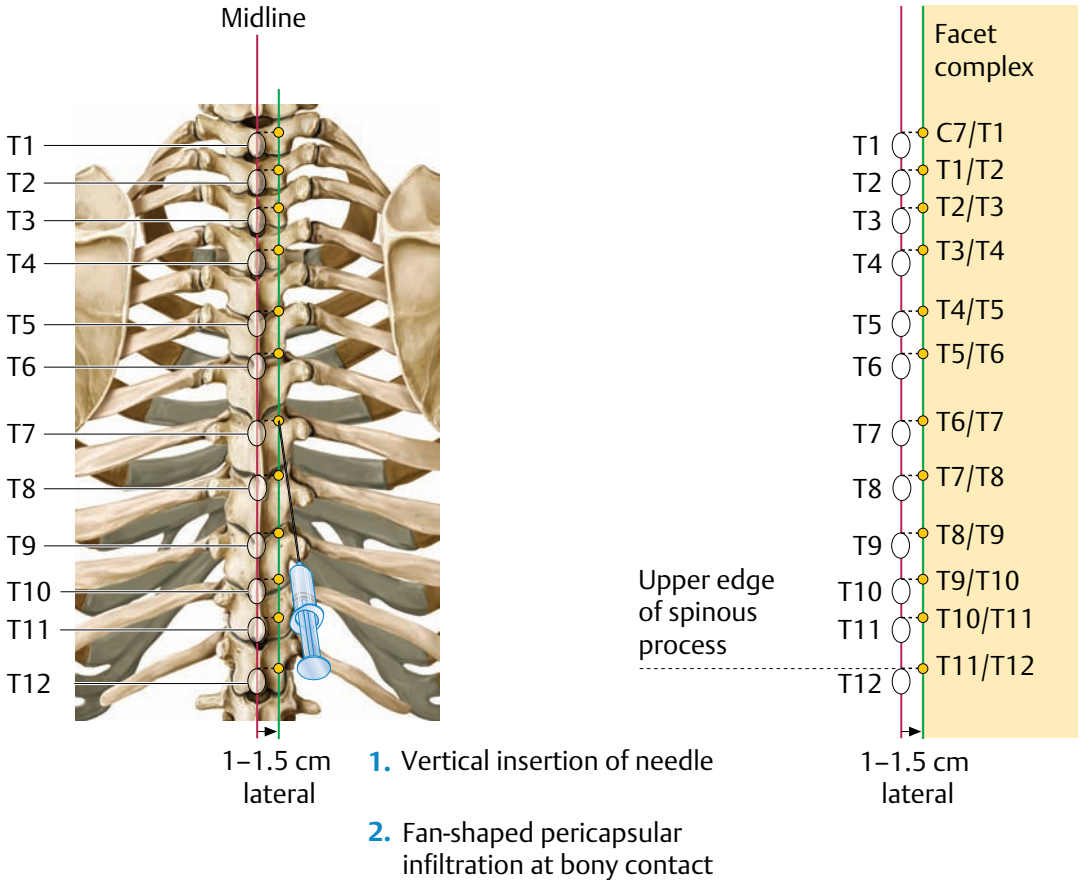


4. Insertion in a 20° caudal direction

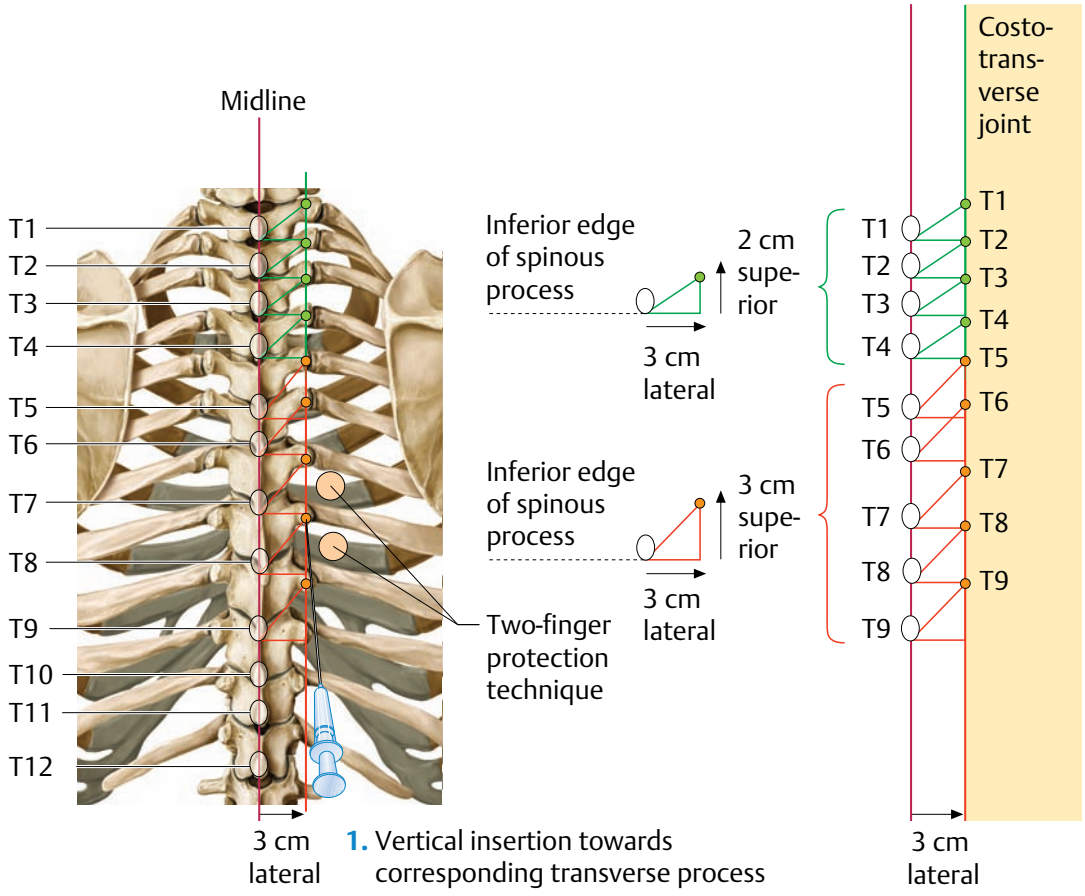


5. 1–2 cm until bony contact is made

TxFI (Thoracic Facet Infiltration)

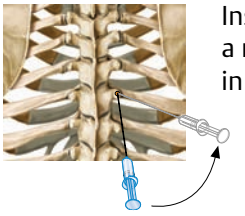


CTB (Costotransverse Block)



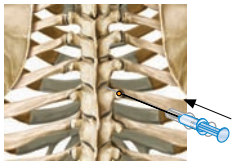
1. Vertical insertion towards corresponding transverse process

2.



Insertion 45–60° in a medial direction in the horizontal plane

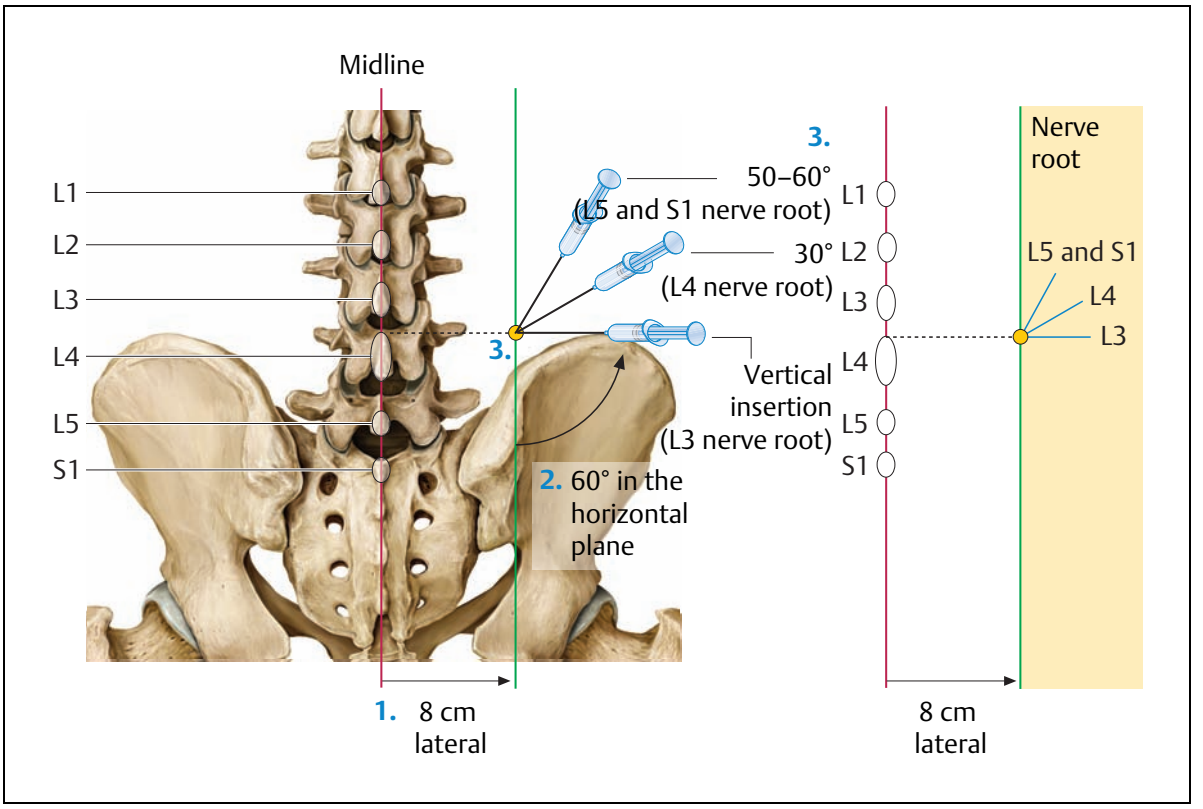
3.



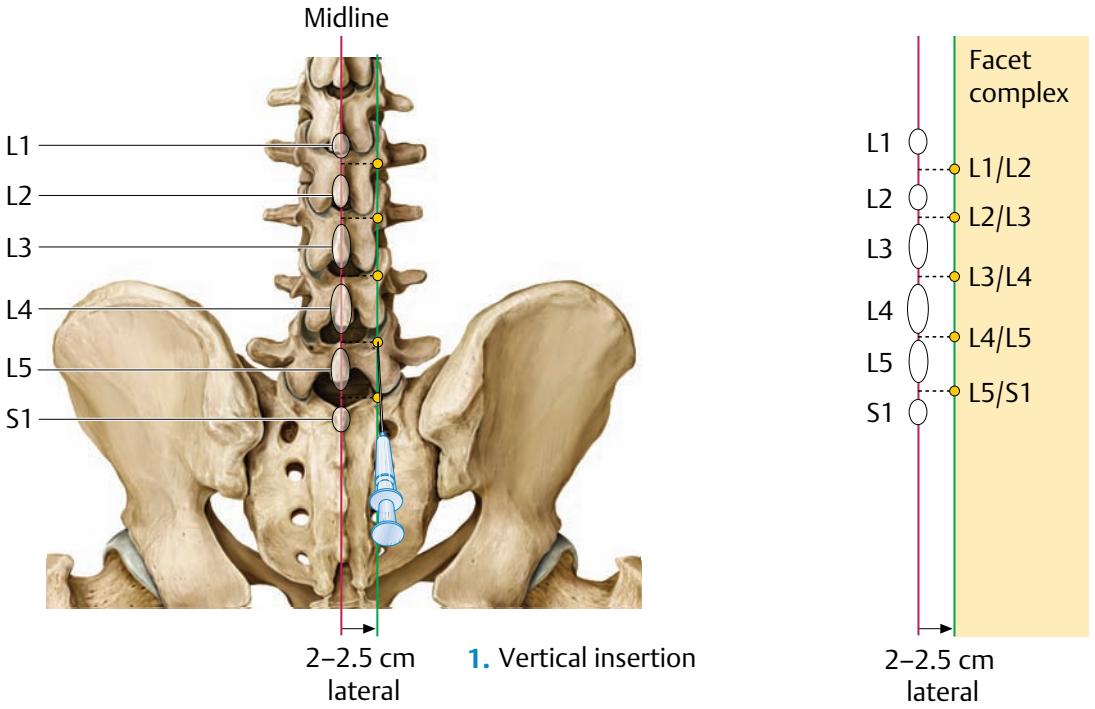
Insertion until bony contact is made (pericapsular infiltration)

Lumbar Spine/Sacrum

LSPA (Lumbar Spinal Nerve Analgesia)

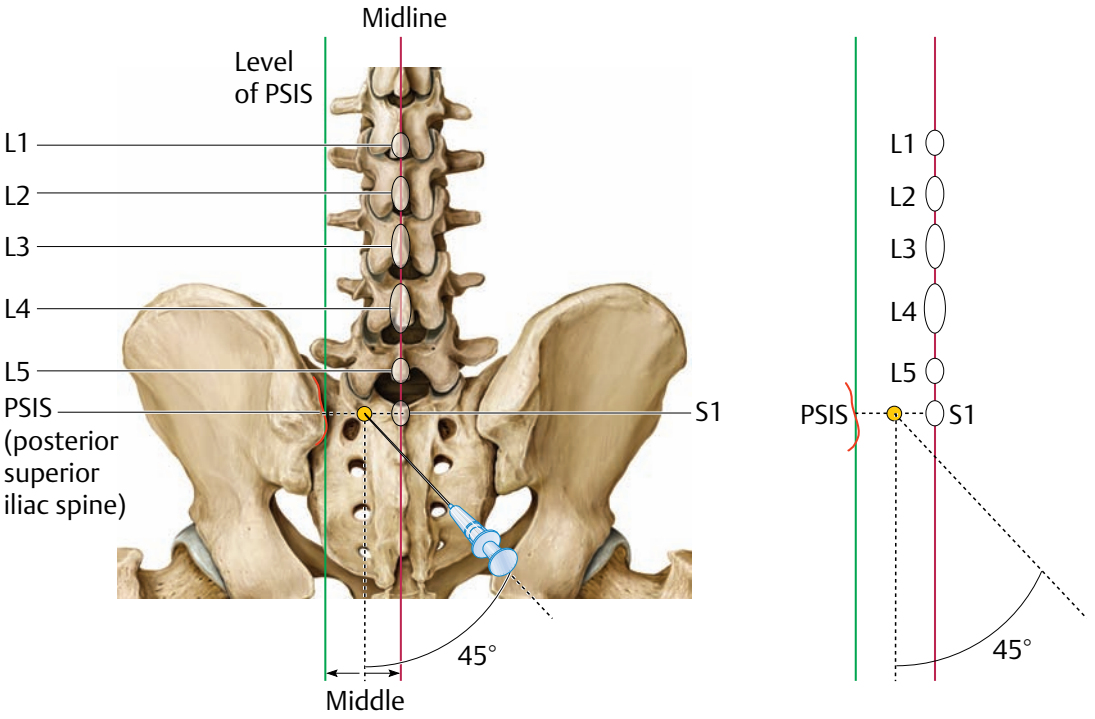


LxFI (Lumbar Facet Infiltration)

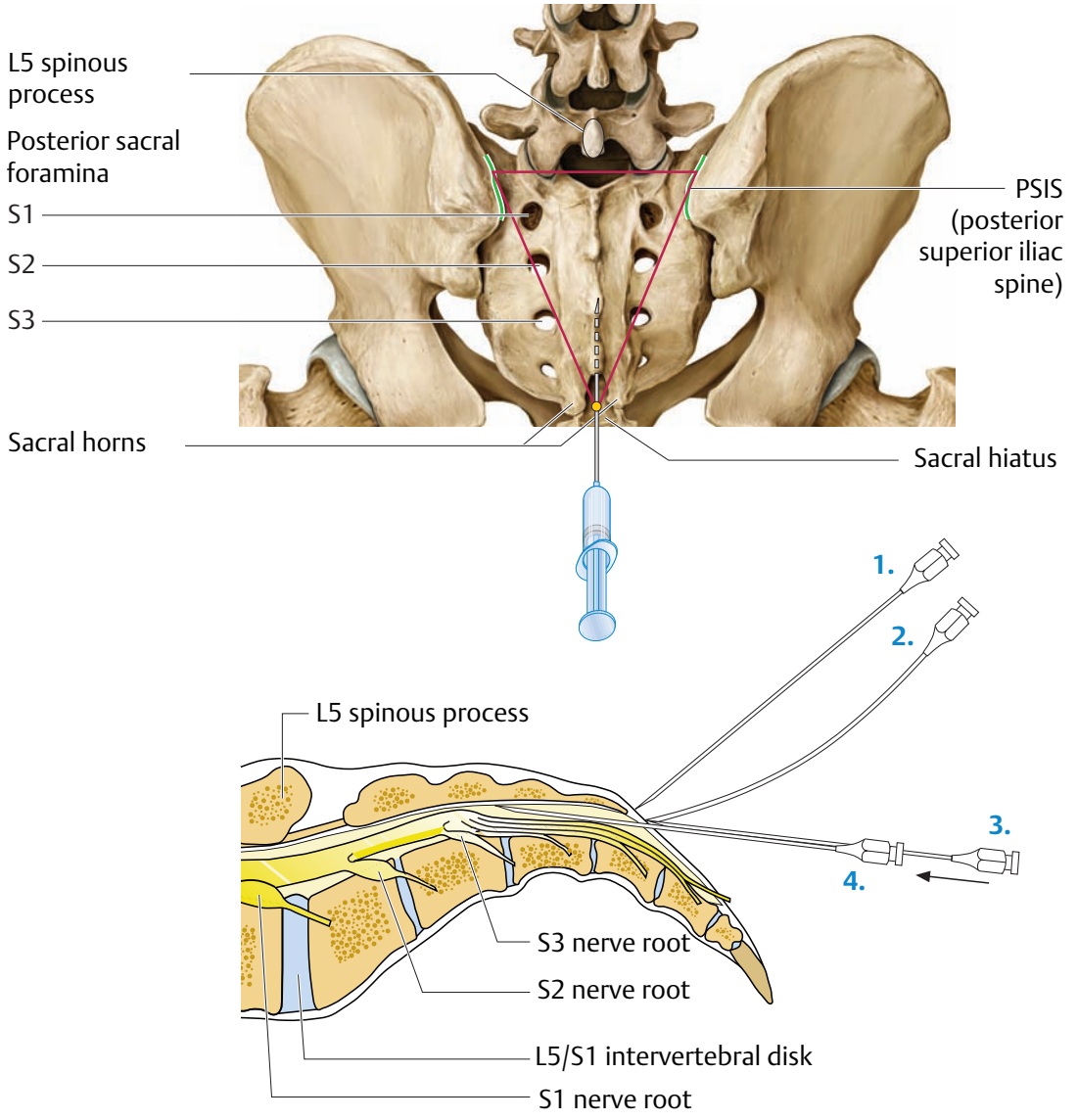


1. Vertical insertion
2. Fan-shaped pericapsular infiltration at bony contact

SIJ Block (Infiltration of Ligaments at the Sacroiliac Joint)

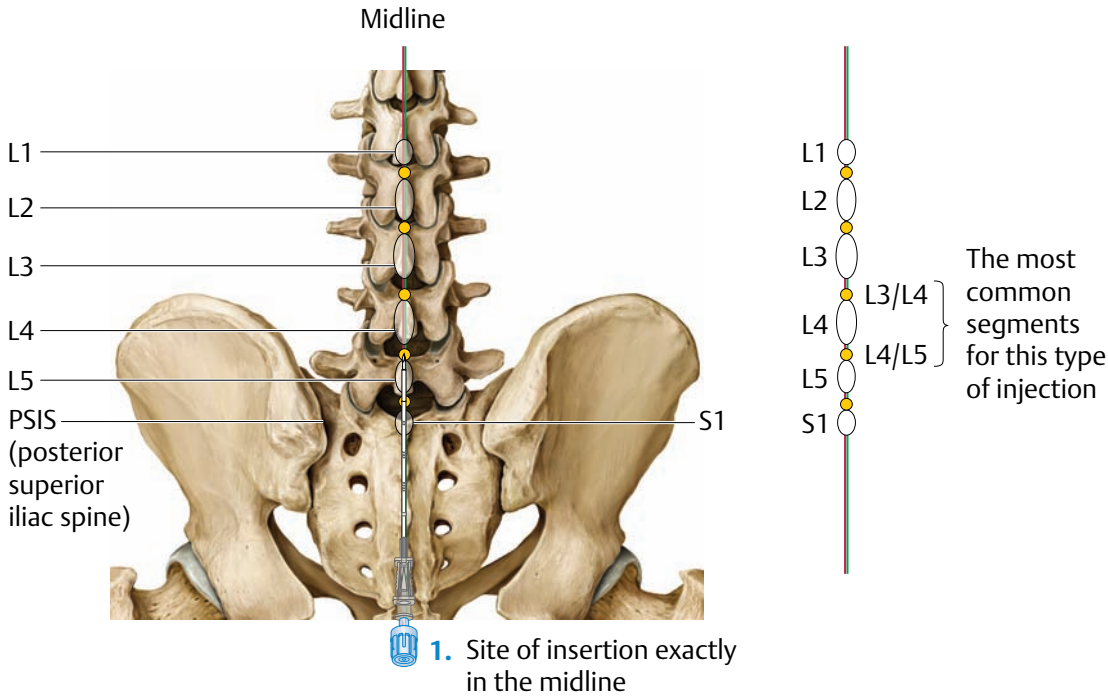


Epi-sacral (Sacral Epidural Injection)

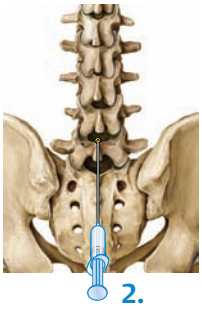


1. Insert the cannula between the sacral horns almost vertical to the skin
2./3. Puncture the ligament (reduction in resistance)
4. Insert the cannula into the sacral canal (up to S3). The cannula shaft forms a continuation of the sacrum.

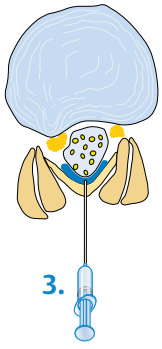
Epi-posterior (Posterior Epidural Injection)



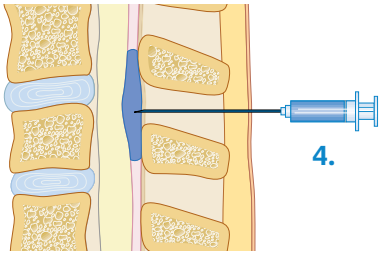
1. Site of insertion exactly in the midline



2.



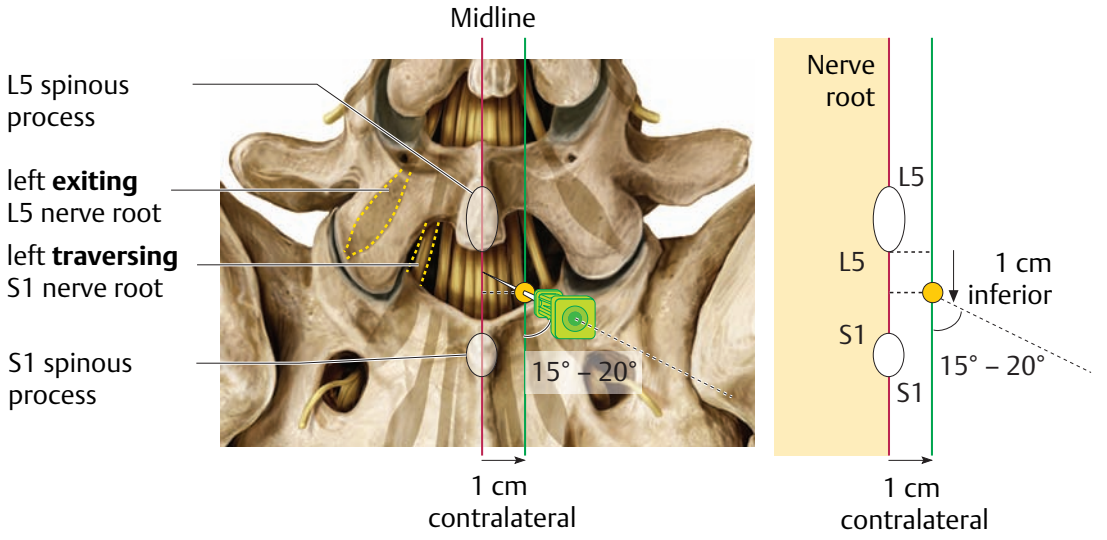
3.



4.

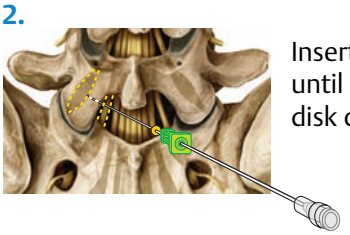
2. – 4. = loss-of-resistence technique

Epi-peri (Epidural Perineural Injection)



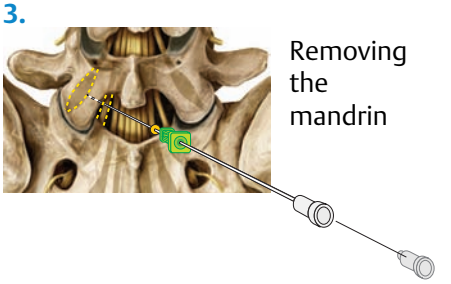
1. L5/S1 interlaminar space. Dura mater removed.
Insertion site for the introducer cannula is 1 cm inferior to the L5 spinous process and 1 cm contralateral at a 15–20° angle. The introducer cannula is inserted through the interspinous ligament until it reaches the ligamentum flavum.

2.



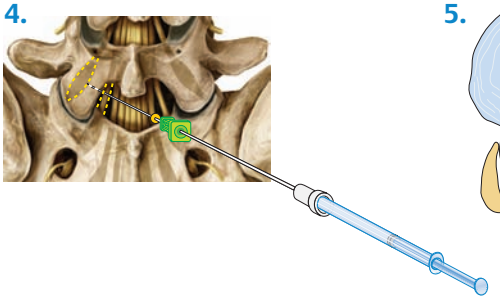
Inserting the 29 G cannula until bony/intervertebral disk contact is made

3.

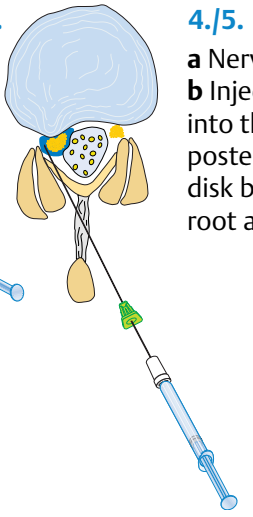


Removing the mandrin

4.



5.



4./5.

a Nerve root anesthesia
b Injection of the suspension (1 mL) into the anterolateral epidural space posterior to the L5/S1 intervertebral disk between the **exiting L5** nerve root and the **traversing S1** nerve root

Conclusion

Injection Therapy of the Cervical Spine

A series of injections of cervical spinal nerve analgesics, complemented by one or two cervical epidural injections, is the focus of the injection therapy of the cervical spine. Facet infiltrations, occipital nerve infiltrations, and trigger point infiltrations are added to this treatment program according to the indications and the presenting symptoms.

Injection treatment of the cervical spine entails a higher risk than injections in other sections of the vertebral column because of its location close to the proximal spinal cord and pleura. It is therefore advisable for the procedures to be radiographically monitored, as is the case for cervical epidural injections. Despite the many possible complications, injection therapy at the cervical spine is an alternative to complex decompression surgery, even more so than in the lumbar spine, as cervical decompression surgery is always accompanied by fusion or intervertebral disk replacement at the affected cervical spine segment.

NOTE

As long as there are no absolute indications for surgery, injection therapy should be tried before operating on the cervical spine.

Injection Therapy of the Thoracic Spine

Injections into the thoracic spine involve the posterior sections of the thoracic vertebral motor segments. A thoracic intervertebral disk prolapse (extremely rare) cannot be reached with injections. It must be operated on when clinical symptoms and spinal cord compression are present.

In the posterior section of the thoracic vertebral motor segments, degenerative disorders of form and function can lead to the irritation of nociceptors in the intervertebral joints, the costotransverse joints, and the spinal nerves at the thoracic intervertebral foramen. Correspondingly, thoracic facet blocks, costotransverse blocks, and thoracic spinal nerve analgesia are available. These blocks have the following special features:

- Nerve blocks are only rarely required in this area as thoracic syndromes are uncommon.
- Even experienced practitioners do not have as much practice with this type of injection as with injections to the cervical or lumbar spine.
- There is a high risk of pneumothorax.
- There are no useful possibilities for surgical nerve decompression.
- Local and radicular thoracic syndromes have a benign, self-limiting course.
- Image-guided injections do not provide extra safety.

For all these reasons, a routine injection treatment of the thoracic spine is not recommended. Only in cases of persistent thoracic syndromes, which can sometimes be extremely painful, the injection treatment should be carried out by a specialist who is experienced with thoracic joint and nerve root blocks.

NOTE

Injection treatment for the thoracic syndrome is rare and subject to many complications.

Injection Therapy of the Lumbar Spine

The “trouble spot” in the lumbar vertebral motor segment consists of the anterolateral epidural space, the foraminal articular region, and the zygapophyseal joint capsule. All of these can only be reached with an 8–12-cm needle: so-called deep needling. Since the lumbar spine contains no spinal cord, this area is less vulnerable to injections than other sections of the vertebral column, also because of the lack of important neighboring organs. The use of small amounts of anti-inflammatories, generally in the form of cortisone, as well as dilute local anesthetics, additionally reduces the risk. Nevertheless, extreme diligence is advisable to help the patient quickly and to prevent infection, which is the main complication.

The injection treatment mainly targets the compromised nerve root, which is swollen by inflammation. The nerve root is reached via the spinal canal when an epidural injection is used, and as it exits the intervertebral foramen when using spinal nerve analgesia. The epidural–perineural injection is the most effective and safest type of injection, as only the smallest amounts of local anesthetic and anti-inflammatories are required, delivered directly to the source of the compression using the thinnest available needle. This technique is challenging, however, and should first be practiced on an anatomical specimen. It is advisable to use image guidance or CT when learning to check the needle position obtained from palpatory anatomical orientation points. However, if image guidance is used from the very beginning, the physician cannot develop the ability to think in three dimensions, as required for deep needling. Performing injections under image guidance also leads to other problems: the patient is exposed to a large amount of radiation, and the cost factor is also an issue.

NOTE

In cases of lumbar nerve root compression, provided there are no absolute indications for surgery, deep injection therapy should be trialed first before open or percutaneous surgical decompression is attempted.

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